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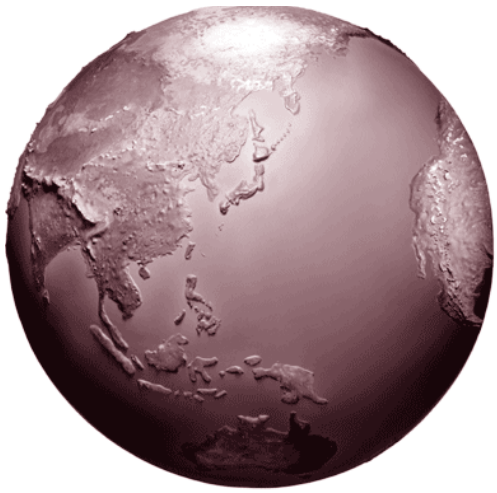
HEI Communication 18

HEALTH EFFECTS INSTITUTE

Household Air Pollution and Noncommunicable Disease

HEI Household Air Pollution Working Group



Household Air Pollution and Noncommunicable Disease

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Communication 18
Health Effects Institute
Boston, Massachusetts

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ABOUT HEI

The Health Effects Institute is a nonprofit corporation chartered in 1980 as an independent research organization to provide high-quality, impartial, and relevant science on the effects of air pollution on health. To accomplish its mission, the institute

- Identifies the highest-priority areas for health effects research;
- Competitively funds and oversees research projects;
- Provides intensive independent review of HEI-supported studies and related research;
- Integrates HEI's research results with those of other institutions into broader evaluations; and
- Communicates the results of HEI's research and analyses to public and private decision makers.

HEI typically receives balanced funding from the U.S. Environmental Protection Agency and the worldwide motor vehicle industry. Frequently, other public and private organizations in the United States and around the world also support major projects or research programs. This document was made possible by funding from Bloomberg Philanthropies. HEI has funded more than 340 research projects in North America, Europe, Asia, and Latin America, the results of which have informed decisions regarding carbon monoxide, air toxics, nitrogen oxides, diesel exhaust, ozone, particulate matter, and other pollutants. These results have appeared in more than 260 comprehensive reports published by HEI, as well as in more than 1,000 articles in the peer-reviewed literature.

HEI's independent Board of Directors consists of leaders in science and policy who are committed to fostering the public–private partnership that is central to the organization. For this report, the final draft was reviewed by independent external peer reviewers, who were selected by HEI for their expertise.

All project results are widely disseminated through HEI's website (*www.healtheffects.org*), printed reports, newsletters and other publications, annual conferences, and presentations to legislative bodies and public agencies.

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Household Air Pollution and Noncommunicable Disease

HEI Household Air Pollution Working Group

INTRODUCTION

Globally, millions of people continue to rely on burning solid fuels to meet their household energy needs, including for cooking, heating, and lighting (Figure 1) (Bonjour et al. 2013; World Health Organization 2015a). As a result, they are often exposed to very high levels of household air pollution (HAP*), a mixture of particles and gases resulting from incomplete combustion of these fuels. In homes where solid fuels are burned in traditional or unimproved stoves, HAP levels have been shown to range widely between studies, with some studies reporting exposures that exceed World Health Organization (WHO) air quality guidelines by several orders of magnitude (Clark et al. 2013; WHO 2014b). In many places, HAP is also a key contributor to ambient air pollution, potentially affecting public health more broadly on national and regional spatial scales (Chafe et al. 2014; Global Burden of Disease from Major Air Pollution Sources [GBD MAPS] Working Group 2016, 2018; HEI 2018).

The very high HAP exposure levels experienced in some parts of the world have led several organizations to estimate the health and economic burden of HAP, identifying it as a major public health concern. In the last five years, as part of their Global Burden of Disease Comparative Risk Assessments, the WHO and the Institute for Health Metrics and Evaluation (IHME) have systematically reviewed the literature on the health effects of HAP exposure. Using the outcomes of these reviews and a number of key assumptions, the WHO estimated 4.3 million deaths globally were attributable to HAP exposure in 2012 (WHO 2014b). The IHME put the estimate for 2016 at 2.6 million deaths; some of these differences are due to reductions in exposure, some to changes in methodology (GBD 2016 Risk Factors Collaborators 2017). The IHME ranked HAP as the 8th leading mortality risk factor globally in 2016 and, combined with ambient air pollution, the leading

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* A list of abbreviations and other terms appears at the end of this volume.

Figure 1. Over a third of the world's population burns solid fuels to meet their household energy needs, including cooking, heating, and lighting. Photo: Ajay Pillarisetti, by permission.

environmental risk factor. It was the 10th leading risk factor for disability-adjusted life-years (DALYs), a measure of the years of healthy life lost.

Most of this health burden is due to noncommunicable diseases (NCDs), those diseases that are not transmittable by infectious agents and that are chronic conditions that develop over time. Worldwide, NCDs constituted 76% of the mortality impacts and 60% of DALYs. The burden created by early mortality and the years of healthy life lost have substantial economic costs for society (World Bank and IHME 2016).

The ongoing major global burden-of-disease (GBD) studies from IHME and WHO, along with other comprehensive reports on HAP and ambient air pollution by the WHO (2014b, 2016), the World Bank (World Bank and IHME 2016), and others, have spurred substantial momentum towards addressing HAP among nongovernmental organizations, intergovernmental organizations (e.g., WHO, World Bank, United Nations Environment Programme, Climate and Clean Air Coalition), and national governments. Yet, in some places HAP has not been as widely accepted as a public health problem among physicians as it has in the environmental community. Reasons for this inconsistency are not clear. For some physicians, it may be the case that the evidence has not yet met scientific standards for guiding decisions in clinical medicine. There may also be a lack of communication and training on the issue within the health sector, particularly in low- and middle-income countries. As a result, decision-making at the national and subnational levels on approaches to address HAP is often led by ministries of energy or the environment, without strong engagement from the ministries of health or the broader health community.

Given the importance of understanding the strength of the evidence among members of the health community, the purpose this report is to provide a critical assessment of the state of the science underlying our understanding of the linkages between HAP and NCDs, while updating previous systematic reviews with the most recently published studies, which are primarily from low- and middle-income countries. The report seeks to answer fundamental questions about the scientific basis for estimating health burden and what the evidence suggests about the exposure reductions necessary to achieve improved health outcomes. The specific objectives are to:

- Update recent systematic reviews of the scientific basis underlying recent estimates of the burden of HAP exposure on NCDs, identify key uncertainties, and evaluate the extent to which the most recent literature has addressed those uncertainties (in the section "Effects of Household Air Pollution on Noncommunicable Diseases");
- Provide perspective on the relative impacts of HAP on the GBD globally and in individual countries and regions around the world (in the section "Burden of Disease Attributable to Household Air Pollution"); and
- Describe the current state of the science in terms of the health benefits that could be achieved by reducing HAP exposures, both from modeling approaches and field studies (in the section "Health Benefits of Reduced Household Air Pollution Exposures").

This report is not intended to make judgments about whether there is adequate evidence that HAP has a large enough impact on health to justify investment in policies that will change exposures. Instead, the report is intended to provide an updated evaluation of the scientific basis that can then be used to inform judgments about addressing potential health risks posed by HAP.

BACKGROUND

GLOBAL EXPOSURES TO HOUSEHOLD AIR POLLUTION

Over a third of the world's population relied on burning solid fuels in their homes for cooking, heating, and lighting in 2016. While the percentage of the world population relying on solid fuels has declined in recent years in favor of other energy sources, millions of people have not yet made this transition (Figure 2). There are strong regional differences in these trends with the highest rates of reliance on solid fuels remaining in low-income countries in eastern, central and western sub-Saharan Africa (HEI 2018).

When solid fuels are burned in traditional or unimproved stoves, often poorly ventilated, a number of potentially hazardous pollutants are emitted. The most commonly measured are carbon monoxide (CO) and particulate matter (PM) (typically, PM$\leq$2.5 μm and PM $\leq$10 μm in aerodynamic diameter [$PM_{2.5}$ and PM_{10}]). The concentrations of PM in homes where solid fuels are burned often far exceed ambient levels as well as guideline levels for protecting health. Figure 3, adapted from Clark and colleagues (2013), summarizes 24-hour area and outdoor PM concentrations, as well as personal PM exposures (PM_{10}, PM_4, and $PM_{2.5}$ in $\mu g/m^3$) reported in selected studies from the WHO Global Household Air Pollution Measurement database (*www.who.int/indooair/health_impacts/databases_iap/en/*).

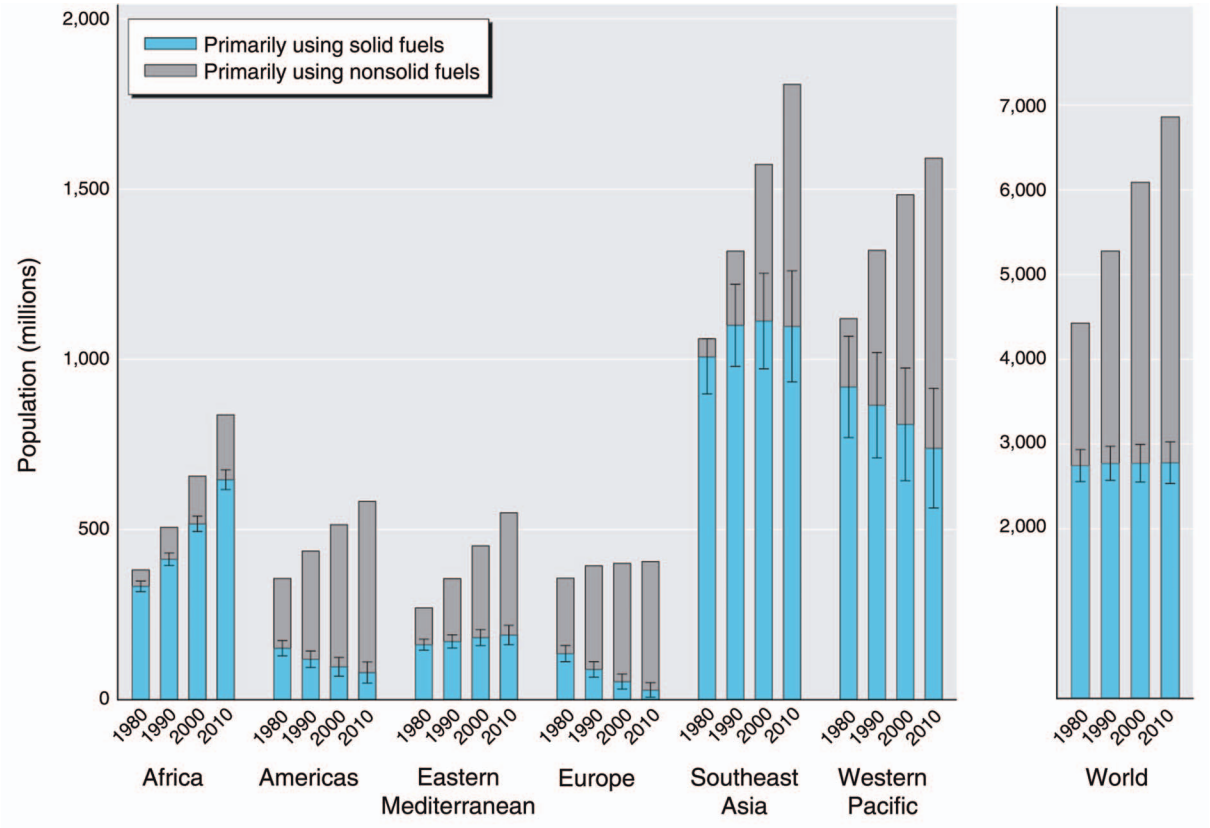

Figure 2. Global and regional trends and 95% confidence intervals in population relying on solid fuels as the main cooking fuel in low- and middle-income countries from 1980 to 2010. Source: Bonjour et al. 2013.

Figure 3 also shows that the WHO Guidelines and first Interim Targets for annual average $PM_{2.5}$ and PM_{10}, the more relevant averaging time for long-term exposures and chronic disease, are exceeded often by orders of magnitude. The guidelines for shorter-term, 24-hour concentrations are also exceeded by substantial margins (see Table 1 for a complete list of WHO guidelines for both annual average and 24-hour average concentrations for $PM_{2.5}$).

BURDEN OF DISEASE FROM NONCOMMUNICABLE DISEASES

NCDs are estimated to account for most of the world's burden of disease from all risk factors (Figure 4). In high-income countries such as the United States and Japan and middle-income countries such as China and India, most deaths and DALYs (predominantly from cardiovascular and respiratory diseases and cancer) are attributable to NCDs. At the same time, DALYs due to communicable diseases such as lower-respiratory infections (LRIs) have markedly declined (GBD 2015 Mortality and Causes of Death Collaborators 2016).

The burden of HAP, through its contributions to the same diseases, is also estimated to be dominated by NCDs. In 2016, NCDs constituted 76% of the mortality impacts and 60% of DALYs (Figure 5) (GBD Compare 2017; IHME 2017). Nearly two-thirds of estimated HAP-attributable deaths from NCDs (47% of the overall total) were from cardiovascular diseases and nearly one-third (23% overall) from chronic respiratory diseases, with the remainder from LRIs and neoplasms (Figure 6). IHME further estimated that, on a global basis, approximately 19% of all DALYs from chronic obstructive pulmonary disease (COPD) and 10% of DALYs from ischemic heart disease (IHD), stroke, and lung cancer were attributable to HAP. The WHO similarly attributed substantial percentages of cardiovascular and respiratory diseases to HAP exposures (Prüss-Üstün et al. 2016).

The estimated burden of HAP exposure from NCDs is in part driven by large-scale health transitions that have occurred over the last decades and that are expected to continue. As life expectancy at birth has increased over the past four decades in most countries, more people are living long enough to develop chronic NCDs, including cardiovascular

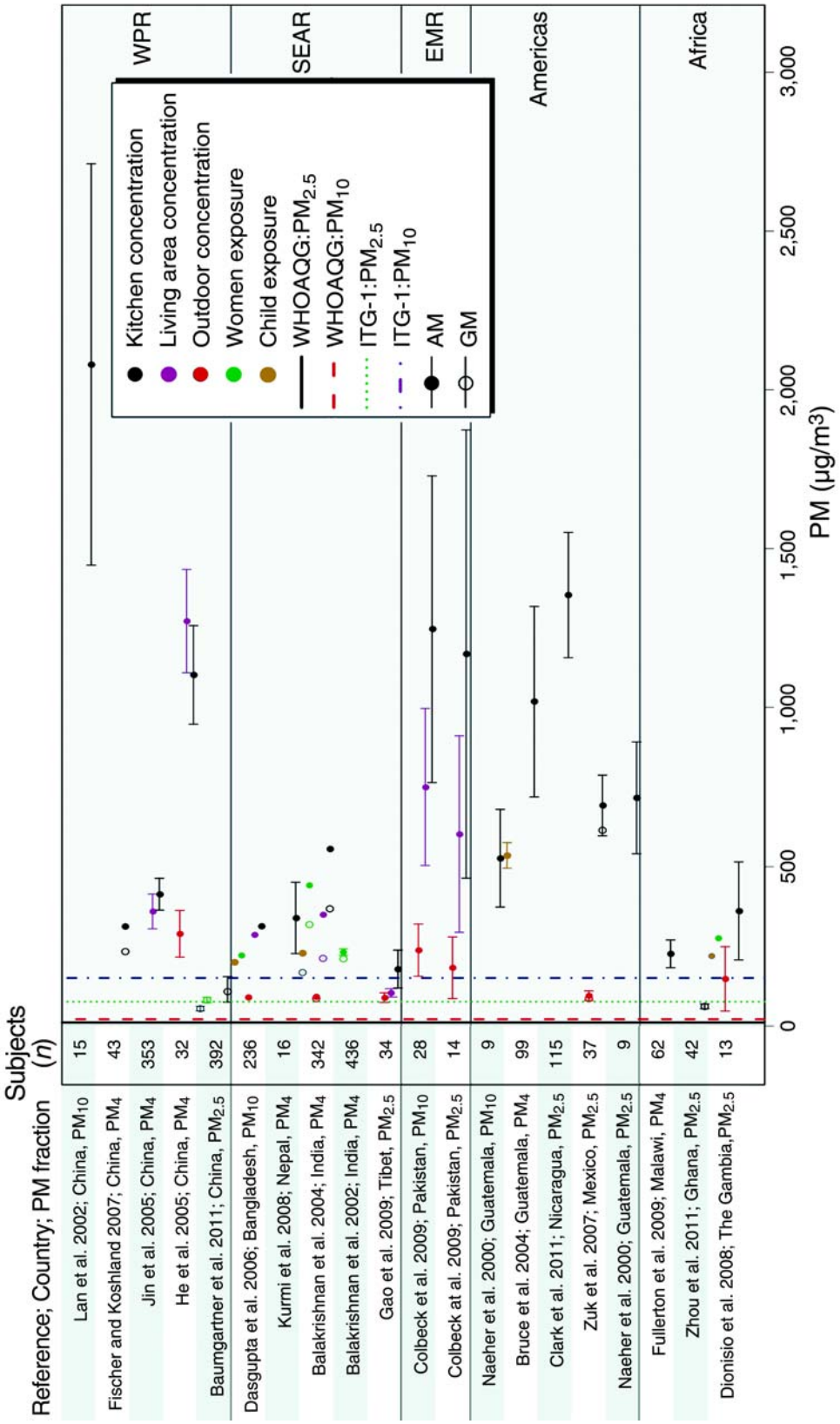

Figure 3. Summary of Global HAP measurements showing both high concentrations and wide variation. Studies are labeled according to the country and reported PM fraction. For some studies that report mean levels across multiple categories, such as season or fuel/kitchen type, results are shown as the pooled means and pooled SDs. Abbreviations: AM = arithmetic mean (filled circle); EMR = Eastern Mediterranean Region; GM = geometric mean (open circle); ITG-1 = interim target guideline 1; PM_4 = PM ≤4 µm in aerodynamic diameter; $PM_{2.5}$ = PM ≤2.5 µm in aerodynamic diameter; PM_{10} = PM ≤10 µm in aerodynamic diameter; SEAR = Southeast Asian Region; WHOAQG = World Health Organization Air Quality Guideline; WPR = Western Pacific Region. Source: Clark et al. 2013.

Table 1. WHO Interim Targets and Guidelines for Annual Average and 24-hour Average $PM_{2.5}$

	Average Value for $PM_{2.5}$ ($\mu g/m^3$)	
Goal[a]	Annual	24-hour
IT-1	35	75
IT-2	25	50
IT-3	15	37.5
Guideline	10	25

Source: World Health Organization 2014b.

[a]IT = interim target.

and respiratory disease (GBD 2015 Mortality and Causes of Death Collaborators 2016; Prüss-Üstün et al. 2016). However, improved medical care and other factors are leading to declines in death rates from COPD, stroke, and other diseases. These dynamics in population health, combined with the transition away from primary household solid fuel use in many countries, have led to a 30% decrease in deaths attributable to HAP from 1990 to 2016 on a global scale (HEI 2018). The burden of disease attributable to HAP remains high, however, particularly in low- and low-middle income countries where a large portion of households still use solid fuels for energy needs. These countries often experience high exposure to both HAP and ambient air pollution, resulting in a "double burden" on public health (HEI 2018).

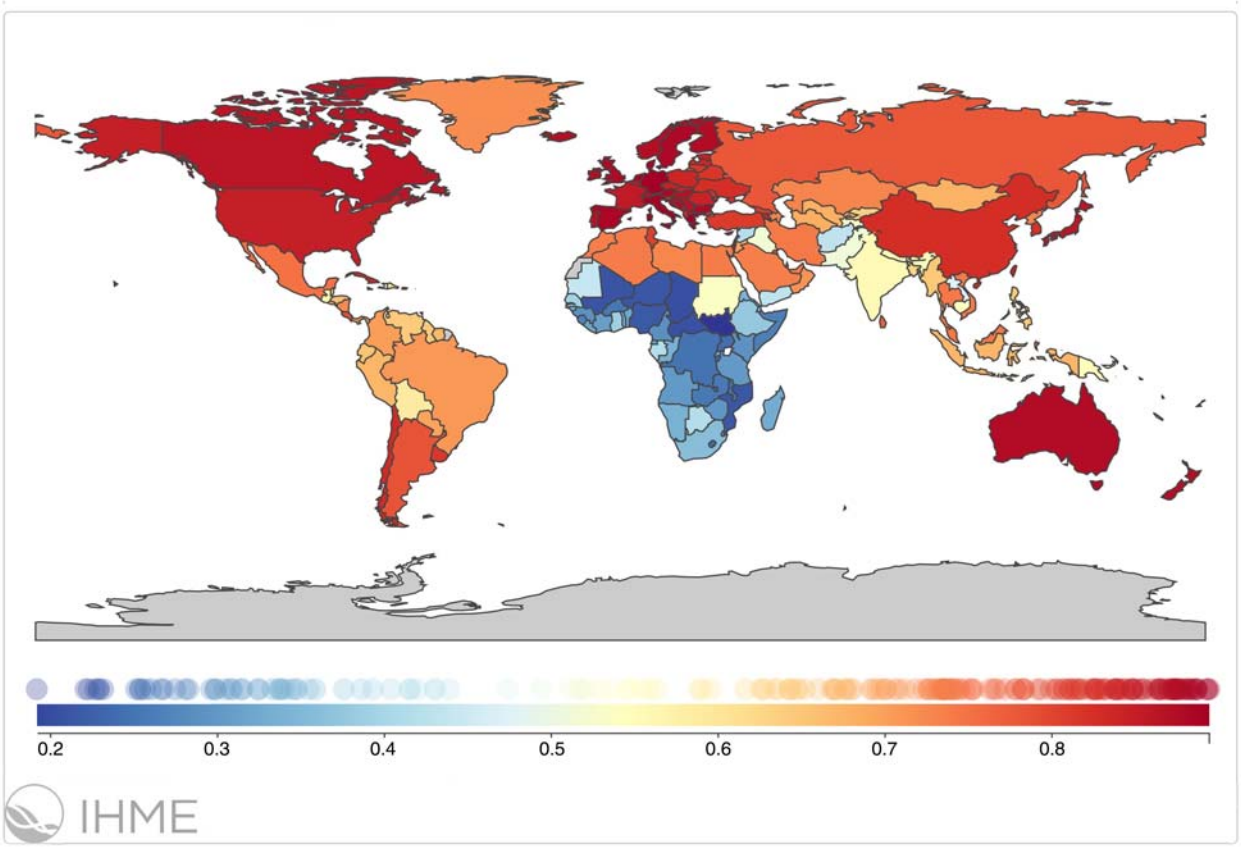

Figure 4. National percentage of total DALYs from NCDs in 2016, both sexes, all ages. Source: IHME 2017.

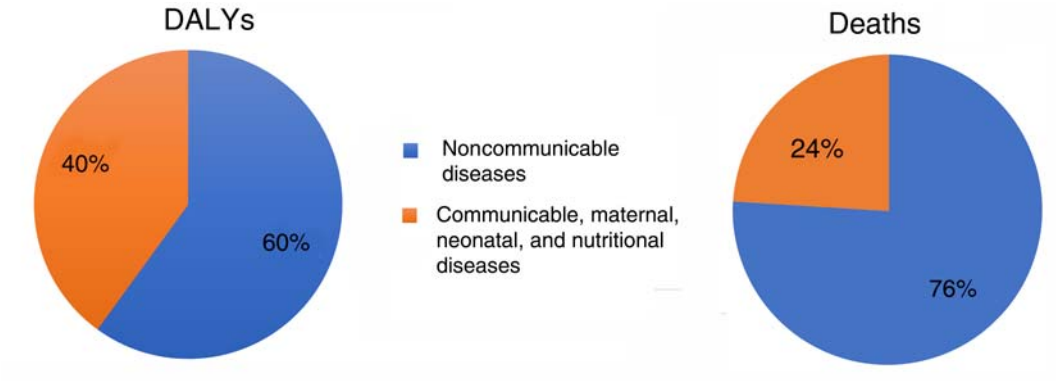

Figure 5. Percentage of burden of disease attributable to HAP globally from NCDs in 2016. Source: IHME 2017.

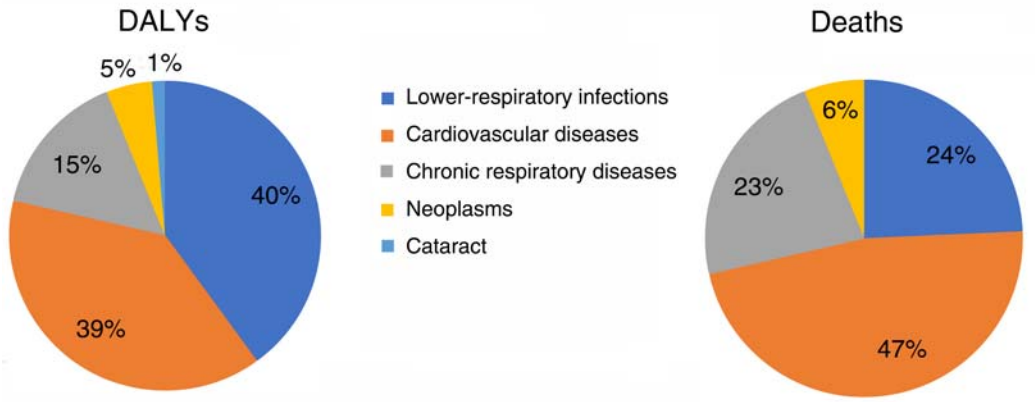

Figure 6. Portion of HAP global burden of disease in 2016 from each health endpoint. Source: IHME 2017.

EFFECTS OF HOUSEHOLD AIR POLLUTION ON NONCOMMUNICABLE DISEASES

What is the scientific evidence underlying the estimates of NCD burden from household air pollution? Over the last few decades, epidemiological studies have been conducted around the world to examine potential linkages between residential solid fuel use and its effects on a range of diseases affecting both adults and children. A number of previous comprehensive reviews of this epidemiological evidence have helped form the basis for the WHO Air Quality Guidelines, the GBD studies, and reports by the World Bank and other organizations. Using the Bradford Hill guidelines for causal evidence (Hill 1965), the WHO Air Quality Guidelines in particular provided detailed assessments of the strength of the evidence for causal associations between HAP and the individual health outcomes that were used to develop their air quality guidelines and interim targets (Table 2) (Bruce et al. 2014). (See Section 2 of Bruce et al. 2014 for tables summarizing the evaluation for each health outcome.)

GBD studies from both the IHME and the WHO have focused on a subset of those outcomes that were judged to have sufficient evidence of a causal relationship to develop exposure–response functions: child LRI, COPD, lung cancer, cardiovascular disease (IHD and stroke), and cataracts (for women only) (Prüss-Üstün et al. 2016; Smith KR et al. 2014). IHME incorporated adult LRIs into its estimates in 2016.

STRUCTURE OF AND APPROACH TO THE CURRENT REVIEW

How strong is the evidence for HAP exposure and NCDs? Given the magnitude of their projected burden and the implications for potential interventions, it is important to understand the strength of the evidence linking HAP exposure with each of these health outcomes. Our objective was to begin with results from previously published literature reviews, rather than conducting a de novo systematic review. We therefore began by assembling the most recent, high quality reviews of the literature for each outcome, including reports from WHO and comprehensive literature reviews of studies examining relationships between HAP and health outcomes published in peer-reviewed journals. We identified 18 previously published reviews of HAP and health outcomes (see Appendix A), coded these according to the outcomes they addressed, and used those that reported quantitative risk values as a starting point for the health outcome sections of this report. While a few reviews addressed multiple health endpoints, most focused on one or a smaller subset; therefore, our assessment for each health outcome begins with a different set of previous reviews.

To update the previous reviews with newly published literature, we searched the literature for new studies of HAP and health outcomes that were published in the peer-reviewed literature after the cut-off dates of those previous assessments. We searched two indexed databases, PubMed and Web of Science, using two *stressor* terms and one *outcome* term in the following format: [("stressor 1" OR "stressor 2") AND "outcome"] (Appendix A). Each search was run without date constraints and then again with date constraints based on the publication of the most recent major review. Additional literature was retrieved from the grey literature using a Google search with a structure similar to that of the indexed database search. Studies were included in the assessment if they remained relevant to the search criteria upon examination and if they reported quantitative risk estimates accompanied by an estimate of precision.

For each health endpoint, we first summarized the results and conclusions from previous assessments. We reported specific evaluations of the strength of the evidence undertaken by the WHO for the recent air quality guidelines, which used Bradford Hill viewpoints (Hill 1965) as a basis for evaluating the strength of causal evidence from epidemiological studies. We then described whether the new studies were or were not consistent with the previous conclusions made by the WHO and others. Key gaps in knowledge are also highlighted. An assessment of the potential biological mechanisms by which HAP influences the incidence or exacerbation of particular diseases was outside the scope of this report.

RESPIRATORY DISEASES

COPD is the third-leading cause of death globally (GBD 2016 Mortality and Causes of Death Collaborators 2017). The WHO conducted its most recent review of studies on

Table 2. Health Outcomes Evaluated in the WHO Recommendation Process[a]

Higher Priority Disease Outcomes Evaluated for WHO Recommendations	Other Disease Outcomes
• *Child acute lower-respiratory infections*	• Adult acute lower-respiratory infections
• Adverse pregnancy outcomes (low birth weight, stillbirth, pre-term birth)	• Child cognitive development
• Stunting	• Asthma
• All-cause child mortality (under 5 years)	• Cancer of the upper aero-digestive tract
• *Chronic obstructive pulmonary disease*	• Cancer of the uterine cervix
• *Lung cancer*	• Tuberculosis
• *Cardiovascular disease*	
• *Cataract*	

Source: Bruce et al. 2014.

[a] Italics = health outcomes that were included in estimates of the global burden of disease from HAP conducted by both the Institute for Health Metrics and Evaluation and WHO (Prüss-Üstün et al. 2016; Smith KR et al. 2014).

COPD's relationship with HAP using Bradford Hill guidelines to judge the strength of causal relationships (WHO 2014b). The authors concluded that the evidence "supports a good case for the relationship between HAP and COPD being causal, with evidence clearest for women." The American Thoracic Society reached a similar conclusion in an earlier 2010 review, writing "there is sufficient evidence of an association between burning of biomass fuel and the development of COPD in women" (Eisner et al. 2010). Eisner and colleagues (2010) cited experimental evidence showing biological plausibility and evidence of exposure–response, including multiple studies that have consistently linked biomass smoke exposure with chronic bronchitis and COPD defined by spirometry. The America Thoracic Society statement also says that evidence is inadequate to infer the presence or absence of a causal relationship in men because they are typically not exposed at high levels over a long period; men have also not been systematically studied.

While numerous individual studies have found associations between HAP exposure and COPD, the conclusions quoted above mask two widespread weaknesses with the literature. First, the literature exhibits a high level of heterogeneity in effect sizes found across studies. Second, systematic reviews have found clear evidence of publication bias. These weaknesses are addressed in more detail later.

The evidence linking HAP exposures to COPD draws largely on case–control and cross-sectional studies, but three additional strands of evidence are available. First, evidence from tobacco smoking — a form of biomass smoke exposure — points to a likely role for HAP in COPD; tobacco smoke is a very well-established risk factor for COPD, and HAP likely affects the same molecular processes as tobacco smoke (Pauwels and Rabe 2004). Second, limited randomized control trials, in which clean cookstove interventions reduced HAP exposures, have largely failed to tie those exposure reductions to slower declines in lung function (measured as decline in forced expiratory volume during the first second [FEV_1] in adult women [Romieu et al. 2009; Smith-Sivertsen et al. 2009]). The Mexico study did find a significant improvement in older women who had good adherence to the intervention (a decline of 31 mL compared with a previous decline of 62 mL over 1 year, $P = 0.01$) but no effect in the intention-to-treat analysis (Romieu et al. 2009). Third, several studies have linked early life ambient air pollution exposures to reduced lung function in children (Gauderman et al. 2015). These reductions in lung function may set the stage for lifetime COPD risk; however, this relationship is the subject of ongoing research but has not yet been established for HAP.

Several recent literature reviews address the respiratory effects of HAP exposures. These reviews largely draw on the same set of original research and reach broadly consistent conclusions. Three limitations characterize the underlying body of epidemiological studies. First, with the lone exception of the two randomized control trials noted earlier, the studies reviewed are case–control or cross-sectional and are therefore vulnerable to omitted variable bias. Second, many of the studies relied on questionnaires to assess COPD, which is not as reliable as objective lung function tests and other diagnostic measures. Third, nearly all the underlying studies relied on fuel-use history, as opposed to direct measurements of exposure, to determine HAP exposure. This approach to exposure assessment precludes exposure–response analysis; it may also result in misclassification exposure for study subjects, which tends to bias results toward a finding of no association.

- Hu and colleagues (2010) carried out a systematic review and meta-analysis of the effects of biomass smoke exposure on COPD. The systematic review identified 11 cross-sectional studies and four case–control studies. Meta-analysis found an overall odds ratio (OR) of 2.44 (95% confidence interval [CI] = 1.90–3.33) for biomass fuel users (relative to those who cook primarily with cleaner fuels). The authors found significant heterogeneity in the ORs (after stratifying for study design) and found evidence of publication bias when examining the entire body of literature. No evidence of publication bias was found for the studies that focused on women.
- Kurmi and colleagues (2010) reported a similar systematic review and meta-analysis of the effects of HAP on COPD and chronic bronchitis (a form of COPD). For COPD they identified 12 valid studies and reported a combined OR of 2.80 (95% CI = 1.85–4.23), and for chronic bronchitis they reported an OR of 2.32 (1.92–2.80) drawn from 13 studies. Their assessment of publication bias showed mixed results. The combined set of publications showed no evidence of publication bias, but the subset of COPD studies that used objective lung function measures did show substantial publication bias. The review found heterogeneity in the design, outcome measures, and effect estimates across studies. In particular, the I^2 value, a measure of the amount of heterogeneity, or dispersion, in meta-analysis, was (68.9%) and significant.
- Po and colleagues (2011) cast a slightly broader net and reviewed all available evidence linking HAP exposures to respiratory disease in rural women and children. For chronic bronchitis in women, they found an OR of 2.52 (95% CI = 1.88–3.38) and for COPD, an OR of 2.40 (1.47–3.93). They found no evidence of an association

between HAP exposure and asthma in adult women or in children, though few studies were available for their review. Their analysis of publication effects found clear evidence of bias. As with the three reviews described above, they found significant heterogeneity across studies. The I^2 for chronic bronchitis (47.3%) was not significant, but the I^2 for COPD (67.2%) was.

- The WHO reviewed the evidence on a wide range of HAP-sensitive outcomes as part of the 2014 emissions standards process (WHO 2014b). For respiratory disease, they conducted systematic reviews of evidence on COPD and on lung function. They reported a meta-analysis of COPD that drew on a slightly different set of papers than did Kurmi and colleagues (2010) and that reported an OR of 1.94 (95% CI = 1.62–2.33). They found a significant I^2 (85%) and clear evidence of publication bias. The WHO review includes two randomized control trials that assessed adult respiratory outcomes resulting from cookstove interventions (one in Mexico and one in Guatemala). Both found that switching to a cleaner biomass cookstove improved self-reported respiratory symptoms, and, as noted above, the Mexico study found reduced rates of FEV_1 decline in older women who had good adherence to the clean cookstove intervention.
- In contrast with the preceding reviews, which comprise formal systematic reviews, Gordon and colleagues (2014) provide an authoritative narrative review from the Lancet Commission of the available literature linking HAP exposures to respiratory outcomes. The commission concluded that "good evidence is available that exposure to HAP is associated with an increased risk of developing COPD" (Gordon et al. 2014, page 833).

Several studies on the effects of HAP exposures on COPD and adult lung function have been published since the reviews outlined above. In Nepal, Neupane and colleagues (2014) found that biogas users had significantly lower odds of airway obstruction compared to biomass fuel users. Pope and colleagues (2015) provided the first evidence linking lung function measurements and respiratory symptoms to personal HAP exposures. They found that recent exposures, assessed via exhaled CO at the time of the respiratory assessment, predict lower lung function and recent respiratory symptoms (cough, phlegm, wheeze, or chest tightness). Average CO measurements of exposure (measured with diffusion tubes) were not predictive of reduced lung function, however. Although not direct assessments of COPD, these studies provide additional support for the HAP associations with COPD identified in prior systematic reviews. Finally, we note that while this review focuses on NCD, there is emerging evidence that HAP exposure erodes the lung's defenses against infection, leaving it vulnerable to pneumonia and other infections (Rylance et al. 2015).

Recent large-scale studies found conflicting support for the hypothesis that HAP exposure is an important risk factor for COPD. In the Burden of Obstructive Lung Disease (BOLD) project, a pooled analysis of 14 sites around the world found no evidence of elevated risk among biomass and solid fuel users compared to people who used cleaner fuels (Hooper et al. 2012). The BOLD protocol used consistent, high-quality spirometry to assess obstruction, but relied entirely on self-reported fuel-use history to assess HAP exposure. Recent updates from new sites using the BOLD protocol found that biomass fuel use was a significant risk factor for COPD in Malaysia (Loh et al. 2016) and in northern India (Koul et al. 2016), but not in Nigeria (Obaseki et al. 2016). Each of these individual BOLD studies is, however, limited by small sample size. A recent analysis of 18,554 adult BOLD study participants found no association between airflow obstruction and solid fuel use for cooking or heating (Amaral et al. 2018). A separate analysis of 12,396 adult BOLD participants from 13 resource-poor countries found a significant association between exposure to HAP and COPD (OR 1.41 [95% CI = 1.18–1.68]), with a stronger relationship in women than in men (Siddharthan et al. 2018). Finally, a large study from China analyzed risk factors for spirometry-assessed COPD in a cohort of approximately 317,000 never-smokers (Smith M et al. 2014). While they found some evidence of an association between cooking with coal and airflow obstruction, they found no evidence of associations with biomass fuels, nor any dose dependence related to years of HAP exposure. These countervailing results underscore the importance of future research that incorporates both good measurements of HAP exposure and of disease (spirometry) and that employs strong study designs that are not prone to confounding by other risk factors for COPD.

Since the systematic literature review by Po and colleagues (2011), several additional studies examining associations between HAP and asthma have been published. These generally support the hypothesis that HAP exposure is a risk factor for asthma. These papers are summarized in Table 3.

CARDIOVASCULAR DISEASES

IHD is the leading cause of death worldwide (GBD 2016 Mortality and Causes of Death Collaborators 2017), and HAP exposures have been suggested to be a major risk factor for IHD and other cardiovascular disease. However, until very recently, direct epidemiological evidence

Table 3. Results from Major Reviews and New Studies on Household Air Pollution and Asthma

Author / Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Cho et al. Preprint							
	1,026 — Physician-diagnosed asthma *n* = 597 Control without asthma *n* = 429	Honduras: Olancho	Case–control	Prevalence/ ORs	Asthma in children and young adults. physician-assessed cases	Age, sex, measures of socioeconomic status (annual household income, maternal education, urbanization), presence of household smokers. Lung function in participants with asthma adjusted for use of controller medications such as inhaled corticosteroids.	OR for solid fuel cooking — significant risk: 1.80 (95% CI, 1.17, 2.78)
Gaviola et al. 2016							
	2,953	Peru: Lima; Puno, rural; Puno, urban; Tumbes	Cross-sectional regressions	ORs	Adult asthma via spirometry	Adjusted for age, sex, height, living at high altitude, smoking, body mass index, having hypertension, daily use of biomass fuels, family history of asthma, and socioeconomic status.	No effect for biomass fuel exposures
Oluwole et al. 2017a, b							
	1,690	Southwest Nigeria: Aba-Nla, Eruwa, Igbo-Ora	Cross-sectional regressions	Adjusted ORs	Child asthma, ISAAC definitions	Age, sex, body mass index, maternal levels of education, passive tobacco smoke exposure, and pet ownership.	ORs for solid fuel cooking — significant risk for any asthma symptoms: 1.33 (95% CI, 1.05–1.69) and severe asthma: 2.37 (95% CI, 1.16–4.84) but not for possible asthma: 1.22 (95% CI, 0.95–1.56)
Wong et al. 2013							
	512,707	108 centers in 47 countries	Cross-sectional regressions	ORs	Child asthma, ISAAC definitions	Sex, region of the world, language, gross national income, maternal education, parental smoking, and six other subject-specific covariates.	Significant risk for asthma symptoms and diagnosis; e.g., OR for wheeze in the past year: 1.78 (95% CI, 1.51–2.10)

Note: CI = confidence interval; ISAAC = International Study of Asthma and Allergies in Childhood; OR = odds ratio.

linking HAP exposure to cardiovascular disease has been lacking, and the 2014 WHO review concluded that it did not support a causal relationship (WHO 2014b). Indeed, at the time cardiovascular disease was included in the GBD risk estimates for HAP, no high quality epidemiological studies linking exposure to increased cardiovascular disease risk had been published. The rationale for including HAP-related cardiovascular disease in studies of the burden of disease drew on two lines of reasoning.

First, numerous studies have found an association between increased blood pressure and HAP exposure (Fatmi and Coggon 2016; McCracken et al. 2012), including three intervention studies (Alexander et al. 2014, 2017; McCracken et al. 2007). Increased blood pressure is a well-validated and direct approach to assessing cardiovascular disease risk and is among the most important cardiovascular disease risk factors. High-quality evidence suggests that a lowering of blood pressure is associated with a reduction in cardiovascular disease events. A meta-analysis of one million patients reported by Lewington and colleagues (2002) provides a strong rationale for the expectation of significant health effects from lowering blood pressure, even in individuals with normal blood pressure. These authors concluded that a 2 mm Hg reduction from usual systolic blood pressure would confer about a 10% lower risk of stroke and a 7% lower risk of death from IHD. A 5 mm Hg reduction would result in a 40% lower risk of stroke and a 30% lower risk of death from IHD, even in individuals with normal blood pressure.

The second rationale relies on the observation that exposures to airborne PM at levels lower than HAP, as in ambient air pollution, and at levels higher than HAP, as in cigarette smoking, have each been associated with an increased risk of cardiovascular disease (Smith and Peel 2010). Assuming that all particles are of equal toxicity, an elevated risk of cardiovascular disease associated with HAP can be inferred from a modeled exposure–response function fit to the observed data from other particulate sources (Burnett et al. 2014). These are the principles behind the integrated exposure–response (IER) functions discussed in the section *Integrated Exposure–Response Functions*. Direct evidence on the differential toxicity of different particle sources is not available; rather existing evidence is supportive of the hypothesis that PM toxicity is difficult to attribute to any one source or component (Lippmann et al. 2013; Vedal et al. 2013).

The first systematic review of the effects of HAP on cardiovascular disease appeared in 2016 (Fatmi and Coggon 2016) and identified five studies (including one that was only published as a conference abstract) that linked HAP exposure to IHD. Three of these found significantly elevated risk from HAP exposure, with ORs ranging from 2.6 to 4.8, and two found no effect. All five studies used fuel-use history as a proxy for exposure, and definitions of cases varied substantially across studies. Study designs were mixed (two were small case–control studies, two were cohort studies, and one was a cross-sectional survey). As discussed later, a more recent and more refined analysis of one of the studies with null results (the Golestan Cohort Study) found a significant association between kerosene-related HAP exposure and cardiovascular mortality (Mitter et al. 2016).

The 2016 systematic review also considered studies addressing heart rate, vascular pathology, biomarkers of oxidative stress and inflammation, and blood pressure. Evidence on these markers had previously been reviewed by McCracken and colleagues (2012). Blood pressure has been the most frequently studied cardiovascular disease outcome and has generally shown positive associations with HAP exposure. Other risk markers (heart rate, vascular pathology, and biomarkers of oxidative stress and inflammation) generally showed associations that were consistent with a relationship between HAP and cardiovascular risk, but evidence was limited.

Limited new evidence clarifying these relationships have been published since the Fatmi and Coggon study (2016) but five studies warrant mention. Three recent papers found evidence of increased blood pressure in pregnant women, which is particularly significant because hypertensive disease during pregnancy can have adverse impacts on the mother and fetus. The implications of these pregnancy studies for cardiovascular disease more generally is probably limited. In Ghana, researchers estimated an exposure–response relationship mid-pregnancy and found that a 1-ppm increase in exposure to CO was associated with 0.43 mm Hg higher diastolic blood pressure (95% CI = 0.01–0.86) (Quinn AK et al. 2016a). The same group also carried out a study using ambulatory blood pressure monitors and found that peak CO exposure (defined as $\geq$4.1 ppm) in the two hours prior to blood pressure measurement predicted elevations in hourly systolic blood pressure (4.3 mm Hg, 95% CI = 1.1–7.4) and diastolic blood pressure (4.5 mm Hg [1.9–7.2]), as compared to blood pressure following lower CO exposures (Quinn AK et al. 2017). This is the first time that 24-hour ambulatory blood pressure results have been reported. In Nigeria, women who received an ethanol cookstove early in pregnancy had a mean diastolic blood pressure late in pregnancy that was 2.8 mm Hg lower than that of control subjects (Alexander et al. 2017). A recent study conducted in a cohort of over 50,000 adults in rural Iran provides the clearest evidence to date of an association between cardio-

vascular disease burden and exposure to some — but not all — forms of HAP. It is the first study to directly link cardiovascular mortality to household energy, though the relationship was limited to fossil fuels (kerosene and diesel). The study showed that the risk of death from cardiovascular causes was about 11% higher among people who used kerosene or diesel (compared with all other fuels) and about 6% lower among liquefied petroleum gas (LPG) users (Mitter et al. 2016). Wood and dung use was not a significant predictor of cardiovascular mortality. The cohort was carefully managed: follow up for mortality was unusually complete, and a physician panel assigned cause-of-death. Finally, a recent Chinese prospective cohort study (Yu et al. 2018) was conducted with 271,217 participants (~11,000 of whom were resurveyed on average 2.7 years after the baseline survey). Participants provided recall information related to exposure to HAP arising from cooking and heating. The analysis also included questions about ventilation and, in the resample, was able to track participants who had changed fuels. Solid fuel use for cooking was significantly associated with higher risk of cardiovascular (hazard ratio [HR] 1.20 [95% CI = 1.02–1.41]) and all-cause mortality (HR 1.11 [1.03–1.20]). Similar hazard ratios were noted for heating with solid fuels. Participants who switched to clean fuels had lower risks of mortality (cardiovascular and all-cause) when compared with persistent solid fuel users (HR 0.83 [0.80–0.99] and HR 0.91 [0.85–0.96], respectively). Use of vented cookstoves was also associated with lower risk.

LUNG CANCER

Recent estimates of the number of lung cancer deaths attributable to exposure to HAP have varied from 272,000 (WHO 2014a) to 158,000 (IHME 2017) deaths per year worldwide (compared with approximately 100,000 deaths in an earlier estimate for 2010 (Smith KR et al. 2014). Expressed in terms of morbidity and mortality, lung cancer associated with HAP was estimated to have caused 3.7 million DALYs in 2016 (IHME 2017), compared with an earlier estimate of 2.1 million DALYs in 2010 (Smith KR et al. 2014). The WHO estimates that HAP from cooking causes approximately 17% of total adult lung cancer deaths worldwide (WHO 2016), though IHME places this estimate at 9% (IHME 2017). (Note that the variation in these estimates can be explained in part by use of the IER functions from the IHME GBD project in the most recent estimates, whereas earlier estimates used other functions.

There is growing evidence of an association between HAP from household fuel combustion and lung cancer (Gordon et al. 2014; Kurmi et al. 2012; Mortimer et al. 2012; Smith KR et al. 2014). Meta-analyses present strong epidemiological evidence that exposure to indoor coal smoke significantly increases lung cancer risk (Zhang and Smith 2007). The International Agency for Research on Cancer (IARC) classified emissions of indoor combustion of coal as carcinogenic to humans (Group 1) on the basis of sufficient evidence both in humans and in animals (Straif et al. 2006). The carcinogenicity of different types of coal may vary significantly (Gordon et al. 2014). There is some evidence that coal smoke is more carcinogenic than biomass smoke (Smith KR et al. 2014). Documentation for the WHO Indoor Air Quality Guidelines, which recommend against the combustion of unprocessed coal in households, concluded that there is high-quality evidence of carcinogenicity of emissions from household coal use. The WHO concluded that there was moderate-quality evidence of a causal effect for lung cancer, a designation that took inconsistency between study outcomes into account, even within a given region (Mainland China and Taiwan) (WHO 2014b).

In 2010, household biomass emissions were classified by IARC as a probable carcinogen (Group 2A) because epidemiological evidence was more limited at the time (Smith KR et al. 2014). More recent analyses (from 2014 and 2015) of the epidemiological evidence on association between household biomass cooking smoke and lung cancer strongly supported inference of a causal relationship (Bruce et al. 2015a; Smith KR et al. 2014). Assessment of the evidence for the WHO Indoor Air Quality Guidelines indicated that there was moderate-strength evidence for a causal association between lung cancer in men and HAP from biomass burning; studies of the same effect in women received a grading of low, mostly because of heterogeneity among observational studies (WHO 2014b). HAP from burning animal dung may be particularly toxic to humans (Gordon et al. 2014).

A 2014 meta-analysis of 13 studies, performed to assess the evidence base for preparation of the Indoor Air Quality Guidelines [Review 4], examined the association between risk of lung cancer in women and exposure to biomass cooking smoke. This review found an OR of 1.58 (95% CI = 1.08–2.32) when analysis was limited to the 6 studies that clearly compared biomass use to cleaner fuel use (WHO 2014b).

The same 2014 analysis described above identified four studies that were conducted solely with female nonsmokers; the pooled OR for lung cancer from these studies was 1.24 (95% CI = 0.82–1.88) (WHO 2014b). When restricted to the single study that clearly compared biomass use to cleaner fuel use among nonsmokers, the effect estimate was 2.08 (1.06–4.07).

Bruce and colleagues (2015a) conducted a meta-analysis to support IHME's 2010 burden-of-disease assessment; the meta-analysis searched 10 databases in 2012, for "studies of clinically diagnosed or pathologically confirmed lung cancer associated with household biomass use for cooking and/or heating." Fourteen relevant studies were identified. The pooled OR for all included studies was 1.17. The majority of studies did not compare biomass use with clean fuel use. Among those studies that focused on women, and that did contain a clear comparison to a clean fuel, the pooled OR was 1.95; when one study that included kerosene in the clean fuel group was excluded, the pooled OR was 2.33. No publication bias was detected.

A 2016 study, done after the major meta-analyses described above, found that never-smokers in Nepal had an elevated lung cancer risk if exposed to HAP from biomass (1.77 [95% CI = 1.00–3.14]) relative to those that had not been exposed (Raspanti et al. 2016). After stratifying based on smoking status and duration (in years) of reported exposure to HAP, the authors described an exposure–response relationship between increasing duration of exposure to HAP and lung cancer risk. They found an OR of 10.16 when they compared never-smokers who had the highest HAP exposure (>65 years) with never-smokers in the lowest quartile (0–45 years of exposure) (Raspanti et al. 2016).

For the 2010 GBD Comparative Risk Assessment, no distinction was made between coal and biomass smoke when calculating the $PM_{2.5}$ IER function for lung cancer. ORs and relative risks (RRs) for both biomass and coal (Table 4) were used to create the IER that was used in the GBD 2010 calculations (Smith KR et al. 2014).(See further discussion in section "Relating PM Exposure and Risk of Disease").

It is important to note some statistical adjustments that could be important in deriving empirical relationships between HAP and lung cancer. Analyses of the risk for lung cancer associated with exposure to HAP are done either with or without adjusting for tobacco smoking status. Analyses often adjust for chronic respiratory disease, because diseases such as chronic bronchitis, tuberculosis, asthma, and emphysema may increase the probability of developing lung cancer later in life (Zhang and Smith 2007). While some studies argue that these adjustments may result in underestimating the ORs of lung cancer, because some previous lung diseases (e.g., COPD) may be on the intermediate path from exposure to lung cancer, others justify them based on uncertainty about whether HAP causes COPD (Zhang and Smith 2007).

Variation in individual susceptibility may also modify HAP–lung cancer associations. In China's Xuanwei County, the role of particular genotypes and proteins has been investigated in the development of lung cancer among residents using smoky coal. Findings suggest that an individual's susceptibility to lung cancer may be increased by a specific genotype (the glutathione S-transferase 1-null genotype), though the magnitude of effect modification is not clear (Zhang and Smith 2007). Recent work indicates that the genetic risk variants for lung cancers not related to smoking may be distinct from those for smoking-related lung cancers (Hosgood et al. 2015; Quinn AM et al. 2016b; Urman and Hosgood 2016).

As with many other health outcomes discussed in this report, lung cancer may be underdiagnosed or misdiagnosed in low- and middle-income countries, potentially leading to substantial underestimation of the overall lung cancer burden in low- and middle-income countries and therefore the HAP-related burden as well (Gordon et al. 2014).

CATARACTS

Cataracts, a condition in which the lens of the eye becomes increasingly opaque, are the main cause of blindness in adults in developing countries (Smith KR et al. 2014; WHO 2016). Worldwide, approximately 285 million people are visually impaired (West et al. 2013). This includes 39 million people who are blind, about 90% of whom live in developing countries. Cataracts are the leading cause of blindness, causing about half of blindness cases worldwide. Additional causes of blindness include glaucoma, corneal opacities, and trachoma. Blindness affects more women than men.

Among eye diseases, the evidence appears strongest for associations between HAP and cataracts. Though the body of literature is smaller than for other health outcomes, such as respiratory disease and cancer, four systematic reviews have synthesized the literature linking HAP and cataracts (Bruce et al. 2014; Kulkarni et al. 2014; Smith KR et al. 2014; West et al. 2013). All studies reviewed were conducted in a South Asian setting, and despite some heterogeneity amongst the studies, most found an association between exposures to biomass fuels and cataracts. RRs for cataracts found from these studies range from 2.12 to 2.47 for women exposed to HAP compared with lower exposures or use of cleaner fuels (see Table 5). Using these effect estimates for cataracts among women developed for the GBD 2010 Study, approximately 35% of the cataract burden in women and 24% of the total burden of cataracts globally was estimated to be associated with cookstove smoke (Smith KR et al. 2014).

The WHO concluded that the evidence suggests "a reasonable case for causality, although experimental evidence is lacking" (Bruce et al. 2014). The final grading was moderate, reflecting upgrading for large effect estimates and

Table 4. Results from Major Reviews and New Studies on Household Air Pollution and Lung Cancer

Author/ Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Bruce et al. 2015a							
	Systematic review: fourteen eligible studies provided 8,221 cases and 11,342 controls	Systematic review: global	All studies included in systematic review were case–control designs	Pooled ORs	Clinically diagnosed or pathologically confirmed lung cancer	Most studies adjusted for potential confounders: age, sex and active smoking. Two studies made no adjustments. Some adjusted for other potential confounders: socioeconomic status and environmental tobacco smoke.	**Biomass —** Cooking and heating: 1.17 (95% CI, 1.01–1.37) Cooking only: 1.15 (95% CI, 0.97–1.37) Male: 1.21 (95% CI, 1.05–1.39) Female: 1.95 (95% CI, 1.16–3.27)
Hosgood et al. 2011							
	Meta-analysis of 25 case–control studies: 10,142 cases, 13,416 controls	Meta-analysis with a focus on geographic variation: Africa, Europe, North America, India, Mainland China, Taiwan	All studies included were case–control studies	Adjusted ORs	Lung cancer risk	Adjusted for potential confounders of lung cancer, which may have included smoking, age, and socioeconomic status, among others.	**Coal —** Worldwide: 2.15 (95% CI, 1.61–2.89) **Coal—** Mainland China and Taiwan: 2.27 (95% CI, 1.65–3.12)
Hosgood et al. 2015							
	1,731 never-smoking female lung cancer cases, 1,349 never-smoking female controls	Female Lung Cancer Consortium in Asia: Mainland China, Hong Kong, Taiwan, Singapore, Japan, South Korea	Multistage genome-wide nested case–control association study	ORs	Histologically confirmed lung cancer	Models included the main effects of single-nucleotide polymorphism and environmental exposure and their interaction term as well as age, study, and environmental tobacco smoke.	Observed a 20% increased risk of lung cancer associated with solid fuel use 1.2 (95% CI, 1.0–1.4) Observed a 30% increased risk of lung cancer associated with coal use 1.3 (95% CI, 1.0–1.6)

Table continues next page

Table 4 (*Continued*). Results from Major Reviews and New Studies on Household Air Pollution and Lung Cancer

Author /Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Raspanti et al. 2016							
	606 lung cancer cases and 606 healthy controls	Nepal	Case–control	Adjusted ORs	Lung cancer risk	Adjusted for tobacco use, tuberculosis status, socio-economic status, age, gender, ethnicity, and exposure to second hand smoke.	Increased risk of lung cancer among those exposed to HAP from biomass burning 1.77 (95% CI, 1.00–3.14)
Smith KR et al. 2014							
	Coal — Systematic review: 25 eligible studies provided a total of 23,558 cases and controls **Biomass —** Systematic review: 14 eligible studies provided 25 independent estimates	**Coal —** Systematic review: Africa, Europe, North America, Mainland China, Taiwan **Biomass —** Systematic review: India, China, Hong Kong, Taiwan, Singapore, Japan, Europe, USA, Canada, Brazil	**Coal —** All studies included were case–control designs per the meta-analysis inclusion criteria **Biomass —** All study designs eligible for inclusion	**Coal and Biomass —** Adjusted ORs	**Coal and Biomass —** Majority of studies lung cancer defined by histology, otherwise by physician diagnosis with X-ray	**Coal —** Adjustment was carried out for smoking, age, and socioeconomic status, among others. **Biomass —** All but two studies carried out adjustment for major confounders.	**Coal —** Cooking: 1.81 (95% CI, 1.19, 2.76) Female only, cooking[b]: 1.98 (95% CI, 1.16, 3.36) Male only, cooking[b,c]: 1.31 (95% CI, 1.05–1.76 **Biomass —** Overall: 1.18 (95% CI, 1.03–1.35) Female only, cooking: 1.81 (95% CI, 1.07–3.06) Male only, cooking: 1.26 (95% CI, 1.04–1.52)
Zhang et al. 2007							
	Unspecified	China	Designs of selected studies unspecified	Unadjusted and adjusted ORs	Lung cancer	Adjusted ORs were adjusted for smoking status and chronic respiratory disease.	Men and women unadjusted: 1.86 (95% CI, 1.48–2.35) Men and women adjusted[a]: 2.55 (95% CI, 1.58–4.10) Men unadjusted: 1.79 (95% CI, 1.18–2.72) Men adjusted[a]: 1.5 (95% CI, 0.97–2.46) Women unadjusted: 1.17 (95% CI, 1.02–1.35) Women adjusted[a]: 1.94 (95% CI, 1.09–3.47)

Note: OR = odds ratio.

[a] Adjusted for smoking status and chronic respiratory disease.

[b] Restricted to China and Taiwan.

[c] Interpolated using female:male OR ratio from biomass studies.

Table 5. Results from Major Reviews and New Studies on Household Air Pollution and Cataracts

Author/ Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Bruce et al. 2015b							
	Unspecified	Unspecified	All studies included were reviews	ORs	Risk of cataract	Unspecified.	Exposure to all solid fuel: 2.47 (95% CI, 1.63–3.73)
Kulkarni et al. 2014							
	12 studies yielded 13 comparison which pertained to a total of 4,025 cases of cataract and 7,048 controls	All included studies published from India, Bangladesh, Nepal	Case–control	Meta-regression coefficient	Risk of cataract irrespective of the types of cataract	Conducted subgroup meta-analyses and meta-regression using linear meta-regression models to account for known confounders such as age, area of residence, gender distribution, and type of cataract.	Summary effect size of 2.12 (95% CI, 1.61–2.80)
Ravilla et al. 2016							
	7,518	North and South India	Population-based, randomly sampled clusters	Adjusted ORs	Risk of cataract and type of cataract	Age, sex, study center, socioeconomic status, tobacco use, sun exposure, malnutrition, vitamin C deficiency, and diabetes.	For 1 standard deviation increase in years of biomass fuel use and nuclear cataract — Women: 1.28 (95% CI, 1.10–1.48) Men: 1.04 (95% CI, 0.88–1.23)

Table continues next page

Note: OR =odds ratio.

Table 5 (*Continued*). Results from Major Reviews and New Studies on Household Air Pollution and Cataracts

Author/ Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Smith KR et al. 2014							
	Eight studies included in the meta-analysis	India / India-Nepal border	Six studies used a case–control design and two studies used a cross-section design	Pooled relative risk	Risk for cataract	A number of sensitivity analyses were conducted, of which three, active smoking, sex, and diabetes, were of particular relevance.	Pooled relative risk for the eight studies using a random effects model was 2.46 (95% CI, 1.74– 3.50)
West et al. 2013							
	Methodical review of the literature identified 18 relevant study publications documenting 19 studies	Study locations included India, various states; Indonesia; Bangladesh; Nepal; Tanzania; Ethiopia; Burkina Faso; Pakistan; Guatemala	Various study designs included in literature review	No analysis presented	Studies measured a variety of outcomes including cataract, trachoma/trichiasis, blindness and visual impairment, tears while cooking, and sore eyes in the past month	Each study included its own covariates. Literature review did not present analysis.	Quantitative summary not provided

Note: OR indicates odds ratio.

analogous evidence from smoking studies but downgrading for inconsistency in underlying studies, partly because all of the studies were conducted in the same region. This conclusion was judged to apply only to women, and that stronger data were needed to sufficiently demonstrate linkages for men. Several of the studies reviewed provided separate estimates for men and women, and most either excluded smokers or adjusted for smoking. However, ultraviolet light exposure and diabetes have not been sufficiently explored to understand the degree to which they confound or modify relationships between HAP exposure and the development of cataracts. Ultraviolet light exposure is associated with cataracts and is also greater at higher elevations in several countries where household burning of solid fuel is more prevalent. Two studies controlling for diabetes resulted in a higher summary RR as compared to both those studies that did not control for diabetes and those studies that excluded individuals with diabetes from their study populations. The higher RR that was found when controlling for diabetes could result from other sources of heterogeneity; an additional complexity is that diabetes acts as an intermediate step in the causal pathway between HAP and cataract development. Additional research is needed to improve understanding about these relationships and potential confounders. Despite these gaps in knowledge and a body of literature linking HAP with cataracts that is both relatively small and limited to South Asia, the evidence was judged strong enough to make the case for causality.

More recently, a major population-based study in India appears to provide additional support for an association of biomass fuel burning with cataracts in women (Ravilla et al. 2016). The adjusted OR for a one standard deviation increase in years of biomass fuel use and nuclear cataract development was 1.28 (95% CI = 1.10–1.48) in women and 1.04 (0.88–1.23) in men. This estimate adjusted for age, study center, socioeconomic status, tobacco use, sun exposure, malnutrition, vitamin C deficiency, and diabetes. Kerosene use was also associated with cataracts in women but not in men. This study strengthens the evidence base for an association between HAP and cataracts for women in South Asia, particularly given the consideration of several key confounders that were not included in previous studies. Importantly, the RR estimate for nuclear cataract development in women was substantially lower than the RRs given by the previous reviews for cataracts as a whole, which could have substantial implications for HAP-attributable burden estimates. In addition, evidence for associations between HAP and cataract development outside of South Asia and in men require further exploration to establish associations with confidence.

Additional evidence exists for potential relationships between HAP and trachoma, blindness, and general visual impairment, but the evidence base is too limited currently to draw conclusions about the strength of these associations (West et al. 2013). Self-reported eye irritation and "tears when cooking" are often cited as complaints in studies of the health effects of solid fuel cooking, and results from interventions in Guatemala and Pakistan indicate that these symptoms may be reversible with improved stoves that lower exposure levels (Díaz et al. 2007; Khushk et al. 2005).

OTHER HEALTH EFFECTS

Emerging evidence indicates that HAP is also associated with a range of additional health outcomes, including low birth weight and other birth outcomes, cognitive and neurological effects, diabetes, and cervical cancer. Additional evidence pointing to associations between combustion particles and these health outcomes comes from studies on ambient air pollution, cigarette smoking, and environmental tobacco smoke. These health outcomes have not yet been included in the IHME or WHO GBD assessments but may be included in future updates if the strength of the evidence is ultimately deemed sufficient for inclusion.

Birth Outcomes

Several studies have extensively reviewed the literature linking HAP exposure with adverse birth outcomes. Adverse birth outcomes include stillbirth, preterm birth, low birth weight, and stunting. Many of these adverse birth outcomes are most prevalent in low- and middle-income countries, where populations are often exposed to high HAP exposures. For example, of the approximately 2.65 million stillbirths globally each year, about 98% occur in low- and middle-income countries (Bhutta et al. 2011). The previous WHO and IHME GBD studies have concluded that more research is needed to further elucidate the relationship between HAP exposure and birth outcomes (Smith KR et al. 2014; WHO 2016).

Systematic reviews of studies that evaluate relationships between HAP and adverse birth outcomes have focused primarily on stillbirth and low birth weight (Figure 7). Three meta-analyses of studies from multiple countries found strong associations for stillbirths and low birth weight among populations cooking with biomass compared to using clean fuels (i.e. electricity or gas). Pooled RR central estimates ranged from 1.29–1.51 for stillbirths and 1.35–1.40 for low birth weight (Table 6). New evidence provides added support for these conclusions, with an elevated risk of stillbirth for households in India cooking with firewood and kerosene relative to using

electricity and gas (Lakshmi et al. 2013; Wylie et al. 2014). Exposure to some metals (arsenic and cadmium) released from coal combustion for heating have also been associated with decreased birth weight in China, though results are difficult to interpret as some metals had significant associations with decreased birth weight during the heating season (arsenic, barium) while others had the same effect during the nonheating season (barium, iron, thallium) (Zhang et al. 2016). In addition, this study did not adjust for smoking, which is a potentially important source of exposure both to PM and to metals such as cadmium. Based on the findings of these meta-analyses and of new studies, the evidence points to potentially elevated risk of both stillbirth and low birth weight with HAP exposure. However, the body of evidence remains limited in terms of both number of studies and geographical area covered, and in the consideration of poverty and its related risk factors, as a potential confounder.

Additional evidence has been building for relationships between HAP and preterm birth, stunting, mortality in children under 5 years, perinatal morbidity, fetal thrombotic vasculopathy, gestational duration, and other adverse birth outcomes (Table 6). While some studies find elevated risks of these outcomes associated with HAP exposure, there are too few studies to draw strong conclusions.

A larger body of evidence from exposure to much lower levels of ambient air pollution provides additional support for connections between combustion pollution emissions and adverse birth outcomes, including preterm birth (<37 completed weeks of gestation) (Shah et al. 2011) and low birth weight (Holstius et al. 2012; Rich et al. 2015). Associations between $PM_{2.5}$ exposure and preterm birth have

Figure 7. Exposure to HAP during pregnancy and early life may be associated with a range of adverse birth outcomes, including pre-term birth, low birth weight, stunting, and mortality among children under 5 years of age. © Bill & Melinda Gates Foundation/Prashant Panjiar. Used by permission of the Gates Foundation.

Table 6. Results from Major Reviews and New Studies on Household Air Pollution and Birth Outcomes

Author / Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Amegah et al. 2014	Systematic meta-analysis: 19 studies satisfied inclusion criteria	Guatemala, Zimbabwe, India, Southern Pakistan, Peru, Serbia, Gaza Strip, China, Ghana	Cross-sectional—prospective cohort; retrospective cohort; matched case–control; randomized case–control trial	Summary-effect estimates and their corresponding 95% CI	Stillbirth	All but two studies adjusted for a range of potential confounders in the analysis including —demographic; household and socioeconomic factors; maternal nutritional; health and lifestyle factors; neonatal characteristics; and second-hand smoke exposure.	Household combustion of solid fuels resulted in a 29% increased risk of stillbirth (1.29, 95% CI: 1.18–1.41).
Amegah et al. 2014	Systematic meta-analysis: 19 studies satisfied inclusion criteria	Guatemala, Zimbabwe, India, Southern Pakistan, Peru, Serbia, Gaza Strip, China, Ghana	Cross-sectional—prospective cohort; retrospective cohort; matched case–control; randomized case–control trial	Summary-effect estimates and their corresponding 95% CI	Low birth weight	All but two studies adjusted for a range of potential confounders in the analysis, including —demographic; household and socioeconomic factors; maternal nutritional; health and lifestyle factors; neonatal characteristics; and second-hand smoke exposure.	Household combustion of solid fuels resulted in a 35% increased risk of low birth weight (1.35, 95% CI, 1.23–1.48).
Bruce et al. 2013	Systematic meta-analysis: 4 studies on stillbirth (no new eligible studies of stillbirth identified in the review update)	India and Pakistan	Observational studies	ORs	Stillbirth	Not specified.	Pooled OR: 1.51 (95% CI, 1.23– 1.85)

(Table continues next page)

Note: CI = confidence interval; EE = effects estimate; OR = odds ratio.

Table 6 (*Continued*). Results from Major Reviews and New Studies on Household Air Pollution and Birth Outcomes

Author / Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Bruce et al. 2013							
	Systematic meta-analysis: 1 study identified with an estimate for preterm birth	Rural India	Cohort study	Adjusted OR	Pre-term birth	Not specified.	OR: 1.43 (95% CI, 1.11–1.85) one study
Bruce et al. 2013, 2015b							
	Systematic meta-analysis: 7 studies on low birth weight	Guatemala, India, Zimbabwe, Pakistan, Gaza Strip	1 randomized control trial and 6 observational studies	Pooled OR	Low birth weight	Not specified.	Pooled OR — All births: 1.40 (95% CI, 1.26–1.54); Pre-term births: 1.51 (95% CI, 1.25–1.83)
Bruce et al. 2013, 2015b							
	Systematic meta-analysis: 4 studies reported on the risk of stunting	Cambodia, Dominican Republic, Haiti, Jordan, Moldova, Namibia, Nepal, India	Cross-sectional/ cohort	Adjusted OR	Stunting	All studies provided adjusted estimates.	OR — Moderate stunting: 1.27 (95% CI, 1.12–1.43); Severe stunting: 1.55 (95% CI, 1.04–2.30)
Bruce et al. 2013; Pope et al. 2010; Smith KR et al. 2014							
	Systematic meta-analysis: 4 studies on stillbirth	Southern Pakistan and India	2 cohort studies, 1 case–control study, 1 cross-sectional study	Pooled effect estimate	Stillbirth	Confounding variables looked for in assessing methodological quality — active smoking; passive smoking; obstetric care; maternal age; parity; socioeconomic status.	Significant pooled effect for cooking with biomass compared to clean fuels (i.e., electricity or gas) — 1.51 (95% CI, 1.23–1.85)

(Table continues next page)

Note: CI = confidence interval; EE = effects estimate; OR = odds ratio.

Table 6 (*Continued*). Results from Major Reviews and New Studies on Household Air Pollution and Birth Outcomes

Author / Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Lakshmi et al. 2013							
	188,917 ever-married women	India	Cross–sectional study	Prevalence ratios	Stillbirth	Place of residence; literacy status (mother and father); religion, source of lighting; standard of living index; antenatal care received during pregnancy; complications during pregnancy; history of previous abortion or stillbirth; premature labor; age at last pregnancy; and gravid status.	Prevalence ratios for cooking with fuel indicated relative to cooking with liquefied petroleum gas or electricity — Firewood: 1.24 (95% CI, 1.08–1.41) Kerosene: 1.36 (95% CI, 1.10–1.67)
Naz et al. 2015							
	18,308 children	Bangladesh	Cross-sectional study	ORs	Under-five mortality	Household wealth index; mother's education; mother's working status; mother's age; wall material of household; place of residence; breastfeeding status; location of kitchen.	HAP was not strongly associated with overall neonatal, infant, or under-five mortality in the context of overall decreasing trends in under-five mortality. Neonatal: 1.49 (95% CI, 1.01–2.22); Infant: 1.27 (95% CI, 0.91–1.77); Under five: 1.14 (95% CI, 0.83–1.55)
Pope et al. 2010							
	Systematic meta-analysis: 6 studies on stillbirth	Guatemala, India, Zimbabwe, Pakistan	2 cross-sectional surveys, 2 cohort studies, 1 case–control study, 1 randomized controlled trial	ORs	Low birth weight	Confounding variables looked for in assessing methodological quality — active and passive smoking; maternal age; parity; socioeconomic status; maternal malnutrition; vitamin supplementation.	Indoor air pollution was associated with 38% increased risk of low birth weight (OR = 1.38, 95% CI, 1.25–1.52) for cooking with biomass compared to clean fuels such as electricity or gas.

(Table continues next page)

Note: CI = confidence interval; EE = effects estimate; OR = odds ratio.

Table 6 (*Continued*). Results from Major Reviews and New Studies on Household Air Pollution and Birth Outcomes

Author / Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Rey-Ares et al. 2016							
	926 households with 695 pregnancies and 1,074 children	Bariloche, Argentina and Temuco, Chile	Retrospective cohort study	ORs	Perinatal morbidity	Relevant variables considered to be potential confounders were added to the models.	Perinatal mortality OR — Temuco: 3.11 (95% CI, 0.86–11.32); Bariloche: 1.41 (95% CI, 0.50–3.97)
Rozi et al. 2016							
	1,275 women	Karachi, Pakistan	Case–control study	ORs	Adverse health outcomes — preterm delivery; low birth weight; stillbirth; low Apgar score	Not specified.	Having no slits in kitchen, which is a proxy indicator for indoor air pollution, 1.90 (95% CI, 1.05–3.43)
Wylie et al. 2014							
	1,744	Central East India	Secondary analysis of data from 2 cross-sectional cohorts	ORs and lower CI estimated using exact logistic regression	Stillbirth	Sociodemographic characteristics.	For cooking with wood compared with gas — Unadjusted OR: 2.71 (0.99, infinity) 2.06 (0.08, infinity)
Wylie et al. 2014							
	1,744	Central East India	Secondary analysis of data from 2 cross-sectional cohorts	ORs and lower CI estimated using exact logistic regression	Pre-term birth	Sociodemographic characteristics.	Cooking with wood — Unadjusted OR: 3.11 (95% CI, 2.12–4.59); Adjusted OR: 2.29 (95% CI, 1.24–4.21)

(Table continues next page)

Note: CI = confidence interval; EE = effects estimate; OR = odds ratio.

Table 6 (*Continued*). Results from Major Reviews and New Studies on Household Air Pollution and Birth Outcomes

Author / Year	Study Population (*n*)	Study Location	Study Design	Statistical Measure	Outcome Measured	Covariates Included	Main Findings
Wylie et al. 2017							
	116 women	Dar es Salaam, Tanzania	Cohort study	Adjusted ORs	Fetal thrombotic vasculopathy	Maternal age; body mass index; secondhand smoke exposure; season of exposure measurement; household asset index.	OR per 1 μg/m^3 increase in $PM_{2.5}$ exposure on the ln-scale: 5.5 (95% CI, 1.1–26.8)
Zhang et al. 2016							
	95 residences	Lanzhou, China	Retrospective case–control	Multivariate linear regression model employed to estimate mean changes in birth weight (grams) and gestational age per one unit increase in concentrations of metals	Low birth weight	Adjusted for maternal age, maternal pre-pregnancy body mass index, preeclampsia, gestational diabetes status.	Maternal exposure to metal indicated, change in birth weight per 1 ng/m^3 increase in concentration — As: during heating season, −1.017 (P = 0.049); Cd: during heating season, −2.72 (P = 0.047); Ba: during nonheating season, 0.329 (P = 0.038); Fe: during nonheating season, 0.118 (P = 0.032); Tl: during nonheating season, 0.82 (P = 0.034)
Zhang et al. 2016							
	95 residences	Lanzhou, China	Retrospective case–control	Multivariate linear regression model employed to estimate mean changes in birth weight and gestational age per one unit increase in concentrations of metals	Gestational weeks	Adjusted for maternal age; maternal pre-pregnancy body mass index; preeclampsia; gestational diabetes status.	Maternal exposure to metal indicated, change in gestational age (weeks) per 1 ng/m^3 increase in concentration — As: heating season, −3.89 (P = 0.044); Cd: heating season, −13.12 (P = 0.008); U: heating season, −2.4967 (P = 0.035); Fe: nonheating season, 0.398 (P = 0.048); Zn: nonheating season, −0.508 (P = 0.043)

Note: CI = confidence interval; EE = effects estimate; OR = odds ratio.

been found in Canada, China, and the United States (Brauer et al. 2008; Ha et al. 2014; Huynh et al. 2006; Parker et al. 2008; Qian et al. 2016). It has now been estimated that maternal exposure to ambient $PM_{2.5}$ maybe associated with 2.7–3.4 million preterm births globally (Malley et al. 2017). Cases of adverse birth outcomes attributable to HAP exposure have not been quantified.

Additional studies are underway to evaluate the effects of HAP on pregnant women, adding to the existing literature on placental growth markers (Dutta et al. 2017) and inflammatory biomarkers among pregnant women (Olopade et al. 2017). Ongoing and recently completed randomized control trials (included the large, multicountry HAPIN trial) are also evaluating the relationship between biomass combustion-related $PM_{2.5}$ exposure and birth outcomes. For example, the Tamil Nadu Air Pollution and Health Effects study in Southern India was designed to assess the associations between exposures to ambient and household air pollution and health effects in children, pregnant women, and other adults in Southern India (Balakrishnan et al. 2015b). This study has since recruited 1,285 pregnant women during the first trimester of pregnancy and found that 10 $\mu g/m^3$ increases in $PM_{2.5}$ exposure during pregnancy were associated with a 4 gram (95% CI = 1.08–6.76) decrease in birthweight.

Cognitive and Neurological Effects

Emerging evidence indicates that HAP exposure may have an effect on cognitive development and neurological conditions. HAP may be particularly important as a risk factor for pediatric neurological outcomes as children can be exposed both during the prenatal period and in early childhood while spending a large percentage of their time around the home. The strongest evidence of the cognitive effects of HAP comes from the Randomized Exposure Study of Pollution Indoors and Respiratory Effects (RESPIRE). This randomized trial birth cohort study in rural highland Guatemala found inverse associations between CO exposure of pregnant women during their third trimester and child neuropsychological performance — including visuospatial integration, short-term memory recall, long-term memory recall, and fine motor performance (Dix-Cooper et al. 2012) — that is, the higher the CO levels, the poorer the measures of cognitive performance. However, the sample size was small (39 mother–child pairs) and the study did not distinguish indoor CO from outdoor CO. Re-analysis of field data collected in Belize, Kenya, Nepal, and American Samoa from 1978–1979 also found negative correlations between open-fire cooking and cognitive performance among children, with the strongest associations at the youngest ages (Munroe and Gauvain 2012). However, the sample size for this study was also small (188 children, not all of which were included in each analysis). Previous evidence shows associations between ambient air pollution and other pollutants in wood smoke — such as polycyclic aromatic hydrocarbons, nitrogen oxides, or black carbon — and IQ and learning deficits among children (Bharadwaj et al. 2017; Morales et al. 2009; Perera et al. 2006; Suades-González et al. 2015; Suglia et al. 2007; Wang et al. 2009). Neurodegenerative diseases occurring later in life, such as dementia, are also of concern (Yin et al. 2016).

Diabetes

Limited evidence suggests that HAP may have an effect on diabetes. However, the role of diabetes in characterizing the health effects of HAP exposure is currently unclear. Diabetes may act as both a confounding factor and potentially a variable along the causal pathway for different outcomes, as discussed in the section on HAP cataracts (Smith KR et al. 2014). HAP exposure may be a risk factor for diabetes itself, as indicated by an elevated risk of diabetes associated with household solid fuel use in Shanghai, China (OR = 2.48; 95% CI = 1.59–3.86); the risk increased with increasing duration of solid fuel use (Lee et al. 2012). Diabetes has also been linked with exposure to ambient $PM_{2.5}$ in North America and Europe, with elevated risks for Type 2 diabetes for both men and women but significant only for women (women OR = 1.14; 95% CI 1.03–1.26; men OR = 1.10; 0.93–1.17) (Eze et al. 2015). These findings add support for a relationship between HAP exposure and diabetes, though the evidence is limited for both HAP and ambient $PM_{2.5}$.

Cervical Cancer

Cervical cancer is the fourth most common cancer in women globally, affecting 528,000 women each year (IARC and WHO 2017). The majority of cases occur in less developed regions. In Eastern and Central Africa, cervical cancer is the most common cancer in women. Nearly all cases of cervical cancer are caused by human papillomaviruses. Other risk factors, including HAP, may play an important role in modifying the risk of cervical cancer among individuals infected with human papillomaviruses. While only a few case–control studies have assessed the relationship between HAP and cervical cancer, the limited evidence available suggests that there could be an association between wood smoke and cervical cancer (Reid et al. 2012). For example, a history of exposure to wood smoke in the kitchen resulted in a higher risk of cervical cancer among women in Colombia and Honduras (Sierra-Torres et al. 2006; Velema et al. 2002). Based on these two studies in

South America and two in Asia, a recent review estimated an OR of 6.46 (95% CI = 3.12–13.36) for the association between HAP from wood and coal combustion and cervical neoplasia (Josyula et al. 2015). Elevated cervical cancer risks for women exposed to HAP appears to differ among individuals, driven in part by metabolic genotype, which can play a role in carcinogen metabolism and repair of DNA damage, as shown in both Colombia and North India (Satinder et al. 2017; Sierra-Torres et al. 2006). Associations between HAP exposure and cervical cancer are further supported by larger studies finding relationships between smoking and cervical cancer (Jiang et al. 2015; Roura et al. 2014), but more evidence is needed to confirm these associations, particularly using prospective study designs and adjusting for confounders, such as poverty and tobacco smoking.

KEY GAPS IN KNOWLEDGE

Despite significant evidence linking exposure to HAP with numerous health effects, a number of critical knowledge gaps remain. Martin and colleagues (2013) offer a thorough review of these gaps, which were investigated and described during expert working group meetings. Topics included cross-cutting social, behavioral, scientific, and health-related issues. We focus on and summarize here a subset of those broader concerns relevant to this monograph. A detailed discussion of issues related to estimating exposure can be found in the section of this report "Health Benefits of Reduced Household Air Pollution Exposures."

Improving the Evidence Base for Associations Between HAP and Noncommunicable Diseases

As evident from the section of this report "Effects of Household Air Pollution on Noncommunicable Diseases," many of the associations between HAP and various health outcomes would benefit from further validation and replication.

- *Respiratory diseases*. Expand the focus to include asthma in children and investigate changes in lung function related to decreased exposure in highly exposed groups over longer time frames. Martin and colleague's (2013) suggestion to replicate the RESPIRE trial in other settings has come to fruition; recent trials have been completed in Ghana, Malawi, and Nepal (though results from the Ghana and Nepal trials are still forthcoming). Nearly all the studies linking HAP exposure to COPD and asthma rely on biomass fuel use as a proxy for exposure. This both precludes exposure–response analysis and can lead to exposure misclassification.
- *Cardiovascular disease*. While Martin and colleagues (2013) suggest longer-term observational and intervention studies to determine the risk of cardiovascular outcomes attributable to HAP exposure, Baumgartner and colleagues (2012) instead focus on performing high-quality, but relatively inexpensive and fast case–control studies of different types of heart disease to establish a firmer baseline understanding of potential associations. The estimates of cardiovascular disease burden suggest that the majority of the burden is borne by men, but to date there are no exposure assessment or blood pressure studies in men. Recent evidence from Yu and colleagues (2018) provides stronger evidence of a relationship between HAP exposure and CVD and will likely be incorporated into future versions of the integrated exposure–response functions in IHME's GBD. Without such empirical evidence of the relationship between HAP exposure and cardiovascular disease, burden estimates based solely on the IER remain uncertain.
- *Cancers*. Martin and colleagues (2013) suggest: (1) looking beyond the risk of lung cancer from HAP to other organ systems; (2) further evaluating the risk of cancer for those exposed to biomass-related HAP; including evaluation of potential exposure–response relationships; and (3) investigating the mechanism of cancer pathogenesis and looking for particular windows of susceptibility during development. Others have identified a need to study the association between lung cancer risk and smoke from solid fuels other than wood and coal (Gordon et al. 2014).
- *Eye disease*. Most of the evidence on associations between HAP and cataract development comes from South Asia; replication is needed in other geographical areas and populations. Further investigation of other eye diseases, including trachoma, acute macular degeneration, diabetic retinopathy, dry eye disease, and other outcomes is also needed.

A significant challenge to obtaining high quality epidemiological evidence for a relationship between HAP and NCDs is the long latency between exposure and chronic health outcome, in particular COPD and lung cancer, but also for cardiovascular diseases and other cancers. The assessment of associations of long-term exposures with chronic health outcomes is further complicated by our relatively poor ability to estimate integrated, lifetime exposure given the typical practice of measuring 24- or 48-hour exposures only a few times throughout a study. Evidence from Guatemala and Mexico — based on measurements of kitchen concentrations of $PM_{2.5}$ — indicate that there is both high day-to-day variability within homes and high variability between homes (Cynthia et al. 2008; McCracken et al. 2009; Pillarisetti 2016). Finally, because many

epidemiological studies of biomass exposure and health outcomes use binary indicators of exposure (e.g., use of biomass for cooking/heating or not) there is a need for more studies that measure actual exposures to biomass cooking smoke and chronic disease outcomes to avoid exposure misclassification and to better understand the exposure–response relationships.

BURDEN OF DISEASE ATTRIBUTABLE TO HOUSEHOLD AIR POLLUTION

The WHO and IHME have each estimated the GBD attributable to HAP in terms of premature mortality (deaths) and DALYs. This section summarizes recent estimates of that burden. It describes exposure–response curves currently used to estimate global HAP-related ill health, how those estimates have changed over time, and how they compare with the burden of disease attributed to other major risk factors.

RELATING PM EXPOSURE AND RISK OF DISEASE

WHO and IHME estimates of the burden of disease attributable to ambient or household air pollution require several inputs: (1) exposure–response functions; (2) regional, national, or subnational estimates of exposure; and (3) country-specific background disease or mortality rates. This subsection includes descriptions of global HAP exposure estimates and exposure–response functions. For information on how background disease data are estimated, see descriptions of the GBD from WHO and IHME (Cohen et al. 2017; GBD 2015 Mortality and Causes of Death Collaborators 2016; GBD 2015 Risk Factors Collaborators 2016; WHO 2014a).

Estimating Global HAP Exposures

Estimation of exposure to HAP for the IHME GBD began by determining the proportion of households using coal, wood, charcoal, dung, and agricultural residues as cooking fuels (GBD 2016 Risk Factors Collaborators 2017; World Bank and IHME 2016). Data on fuel use were extracted from Demographic and Health Surveys, Living Standards Measurement Surveys, Multiple Indicator Cluster Surveys, World Health Surveys, country-specific sources, and from the WHO fuel use database. In 2015, IHME extracted 680 data points from 150 countries (GBD 2016 Risk Factors Collaborators 2017; World Bank and IHME 2016).

Subsequently, use of solid fuels was translated first into indoor $PM_{2.5}$ concentrations and then into exposures for men, women, and children. This mapping relies on HAP indoor $PM_{2.5}$ data from 90 studies in 16 countries. For GBD 2015, in countries with no directly measured data, linear mixed models with random intercepts for country, GBD region, and GBD super-region were utilized to estimate the 24-hour kitchen $PM_{2.5}$ concentrations (GBD 2016 Risk Factors Collaborators 2017; World Bank and IHME 2016). For GBD 2016, a model was created for relating measured $PM_{2.5}$ concentrations to IHME's sociodemographic index (a metric based on average income per person, educational attainment, and total fertility rate) and used to predict exposures for all locations and years (GBD 2016 Risk Factors Collaborators 2017; World Bank and IHME 2016). Finally, indoor concentrations were translated to exposures by applying the ratio of personal exposures to area concentrations based on a subset of seven studies from six countries (GBD 2016 Risk Factors Collaborators 2017; World Bank and IHME 2016). These ratios were modeled separately for men, women, and children based on available time–activity data.

Integrated Exposure–Response Functions

Currently, epidemiological studies of $PM_{2.5}$ and many disease outcomes do not address the full range of exposures experienced by populations around the world. Studies of the high ambient $PM_{2.5}$ concentrations experienced in India and China have been particularly sparse and, with a scant handful of exceptions (most notably the RESPIRE trial in Guatemala [Pope et al. 2015; Smith KR et al. 2011]), are also rare among HAP studies. In response to this knowledge gap, researchers developed IER functions that combine risk estimates from four sources of combustion-related $PM_{2.5}$ (ambient air pollution, HAP, environmental tobacco smoke, and active smoking) (Burnett et al. 2014). These functions thus allow estimation across the range of exposures experienced globally.

Burnett and colleagues (2014) estimated IER functions for five disease outcomes:

- Acute LRI[†] in children
- IHD
- Stroke
- Lung cancer
- COPD

These IER curves have been updated regularly for the 2015 IHME GBD study and will continue undergoing revision as new evidence becomes available. A key feature of the IERs for stroke and IHD is that the shape of these exposure–response curves flattens out at high exposure levels, such that the increase in the excess RR is steeper at lower concentrations

[†] As children do not smoke, the IER for acute LRIs in children omits active smoking. Also, of the five curves, this is the only one to include direct HAP observations (from the RESPIRE trial in Guatemala).

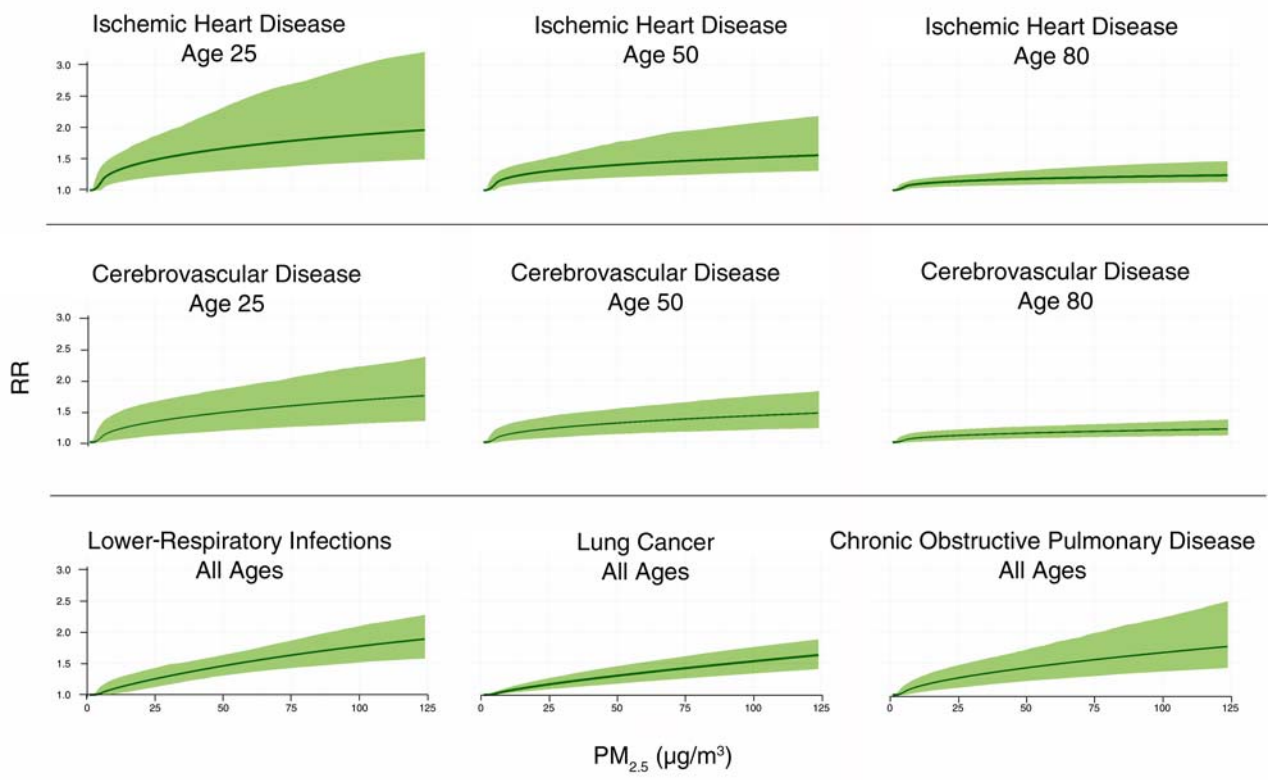

Figure 8. $PM_{2.5}$ integrated exposure–response functions used for the 2015 IHME GBD study. Curves show the central estimate of the integrated exposure–response (solid lines) and their 95% uncertainty intervals (shaded areas). The relative risk equals 1 for $PM_{2.5}$ concentrations of 0–2.4 µg/m^3 (i.e., lower bound of the theoretical minimum risk exposure level uncertainty distribution). Source: Cohen et al. 2017. License: Creative Commons Attribution CC BY 4.0.

than at higher concentrations (Figure 8). The IERs for COPD, lung cancer, and LRIs are more linear than for cardiovascular diseases. Note that $PM_{2.5}$ exposures from HAP often greatly exceed the maximum exposure level depicted in Figure 8, which is 125 µg/m^3. These curves continue to extend beyond 125 µg/m^3 and are anchored on the high end by exposures from active tobacco smoking. For all diseases, the exposure–response curves are monotonically increasing — that is, risk continues to increase across the entire range of exposures.

The nonlinearity of the IERs for cardiovascular diseases implies that major exposure reductions would be needed to achieve substantial health benefits. As discussed previously, evidence linking HAP and cardiovascular disease has been largely lacking, and the data points underlying the IERs for stroke and IHD are all from studies of other sources of exposure to combustion particles (e.g., ambient air pollution). Associations between HAP and cardiovascular disease could take a different shape, with different implications for mitigation approaches, depending on the evidence; future updates of the IER will include evidence from the recent study of Yu and colleagues (2018) on cardiovascular outcomes and HAP exposures in China.

HAP-attributable burden-of-disease estimates are developed using these IERs and the exposure estimates described above, under a hypothetical scenario assuming exposures are reduced to very low "theoretical minimum exposure levels," between 2.4–5.9 µg/m^3. This range is taken from the minimum and fifth percentile of the exposure distributions observed in cohort studies of ambient air pollution that have not necessarily been associated with adverse health outcomes and are lower than the most recent WHO $PM_{2.5}$ annual mean Air Quality Guideline of 10 µg/m^3. Recent evidence from the United States suggests that health effects of $PM_{2.5}$ exposure may occur below the WHO guideline, and that a low-concentration no-response threshold may not exist or be detectable with current methods (Di et al. 2017).

Key Assumptions of and Uncertainties in the IER Approach

As previously mentioned, the IER incorporates exposure and response data from different sources to provide a continuous response relationship representative of the global range of observed exposures. The model requires a number of assumptions, including:

1. Particles from included combustion sources are equitoxic; that is, toxicity varies with the mass of PM inhaled, but not with its composition. This assumption is perhaps the most controversial of those underlying the IERs. Recent evidence is mixed, with a large cohort study suggesting stronger effects on IHD for PM components from coal combustion than others (Thurston et al. 2016), while a recent review (Wyzga and Rohr 2015) indicated no conclusive evidence of differential toxicity. Similarly, the National Particle Component Toxicity studies (Lippmann et al. 2013; Vedal et al. 2013) concluded that there was an absence of evidence that specific PM components could be excluded as contributors to PM toxicity.
2. Disease relationships are a function of long-term daily average exposures and do not depend on the temporal pattern of exposure, which necessarily would vary by source (intermittent active or passive smoking versus population-dependent HAP exposures versus population and location-dependent exposure to ambient air pollution).
3. Exposure to one air pollution source does not interact with exposure to other sources.
4. The form of the relationship between exposure to PM and health outcomes may be nonlinear.

To partially address these limitations — and in acknowledgement that the IER is a model which attempts to fill in evidence gaps — uncertainty in the following inputs is incorporated: the model parameters; the exposure estimates; the counterfactual concentration; and the population attributable risk. The characterization of these uncertainties is described in detail in the supplements to Burnett and colleagues (2014) and Cohen and colleagues (2017).

DISEASE BURDEN FROM HOUSEHOLD AIR POLLUTION GLOBALLY AND IN WORLD REGIONS

Both the WHO and IHME GBD projects estimate the disease burden attributable to HAP as well as to many other environmental, behavioral, and metabolic risk factors like unsafe water, high salt intake, etc. (See comparisons on the IHME GBD website [*https://vizhub.healthdata.org/gbd-compare/*]). Recent estimates by different investigators have attributed between 2.6 million (IHME 2017) and 4.3 million deaths (Prüss-Üstün et al. 2016; Smith KR et al. 2014) attributed to HAP globally each year and approximately 77 million lost DALYs (IHME 2017).

It is important to note that these estimates are largely based on impacts of HAP exposure from cooking and may not include impacts of burning solid fuels or kerosene for other household energy purposes, including heating and lighting. Globally, a large number of what are now considered either as cookstoves or as heating stoves are actually used for both purposes, especially in high latitude or high-altitude regions, or in the wintertime when there is a greater need for space heating (Climate and Clean Air Coalition, International Cryosphere Climate Initiative, Global Alliance for Clean Cookstoves, and Polish Ministry of the Environment 2017a). In low- and middle-income countries, stoves may be primarily designed for cooking, with some of the waste heat used for staying warm. Other stoves may primarily function as heaters throughout the day and night, with heat concentrated towards a cooktop for cooking needs at mealtime. In addition, coal heating stoves are commonly used in places where space heating is required and coal is readily available, including Eastern Europe (e.g., Poland and Moldova) and East Asia (e.g., Mongolia and China) (Climate and Clean Air Coalition, International Cryosphere Climate Initiative, Global Alliance for Clean Cookstoves, Polish Ministry of the Environment, 2017b) (see, for example, Figure 9). Little information is available on how much fuel of different types is burned in different locations and for which purposes. In some cases, particularly where a cookstove is used also as a heater or where solid fuels are burned in separate devices for cooking and heating, households may already be counted in GBD assessments, though these assessments do not break down the portion of HAP and associated health impacts by end use. Given the lack of information on the burden of disease associated with different fuel uses, this section focuses on the available HAP GBD estimates by IHME and WHO, how they compare with other relevant risk factors and how they have changed over time.

Household Air Pollution Burden-of-Disease Estimates and Comparison with Other Risk Factors

For 2016, the IHME GBD project estimated attributable deaths and DALYs for 84 risk factors including ambient and household air pollution using comparable methods (GBD 2016 Risk Factors Collaborators 2017). When assessing deaths caused by all health outcomes, HAP ranked eighth among all risk factors assessed by the GBD project in 2016 (IHME 2017). HAP ranked 10th in the same year when assessed in terms of DALYs caused by all health outcomes. The top three risk factors for all causes of death and DALYs were high blood pressure, smoking, and high

Figure 9. Coal heaters in China. From left to right: Coal heating stove located in a separate room adjacent to the home that transfers heat via radiators; household coal supply; coal heating stove located indoors; coal-burning kang (heated bed). Photos: Ellison Carter, by permission.

fasting plasma glucose. With regard to NCDs specifically, HAP ranked 13th on a global basis (Figure 10). Figure 11 displays the variation across the globe in HAP-attributable death rates from LRIs and NCDs. The regions in which HAP was the highest contributor to NCD deaths are South Asia where it ranked 6th, and sub-Saharan Africa, where it ranked 4th, among all risk factors (Figure 10).

Globally, deaths from NCDs attributable to HAP were stable at approximately 2.5 million per year between 1990 and 2005, decreasing steadily to approximately 2 million deaths in 2016 as shown in Figure 12 (IHME 2017). This trend was largely driven by an increase in attributable NCD deaths in South Asia, a decrease in attributable NCD deaths in East Asia, and smaller increases and decreases

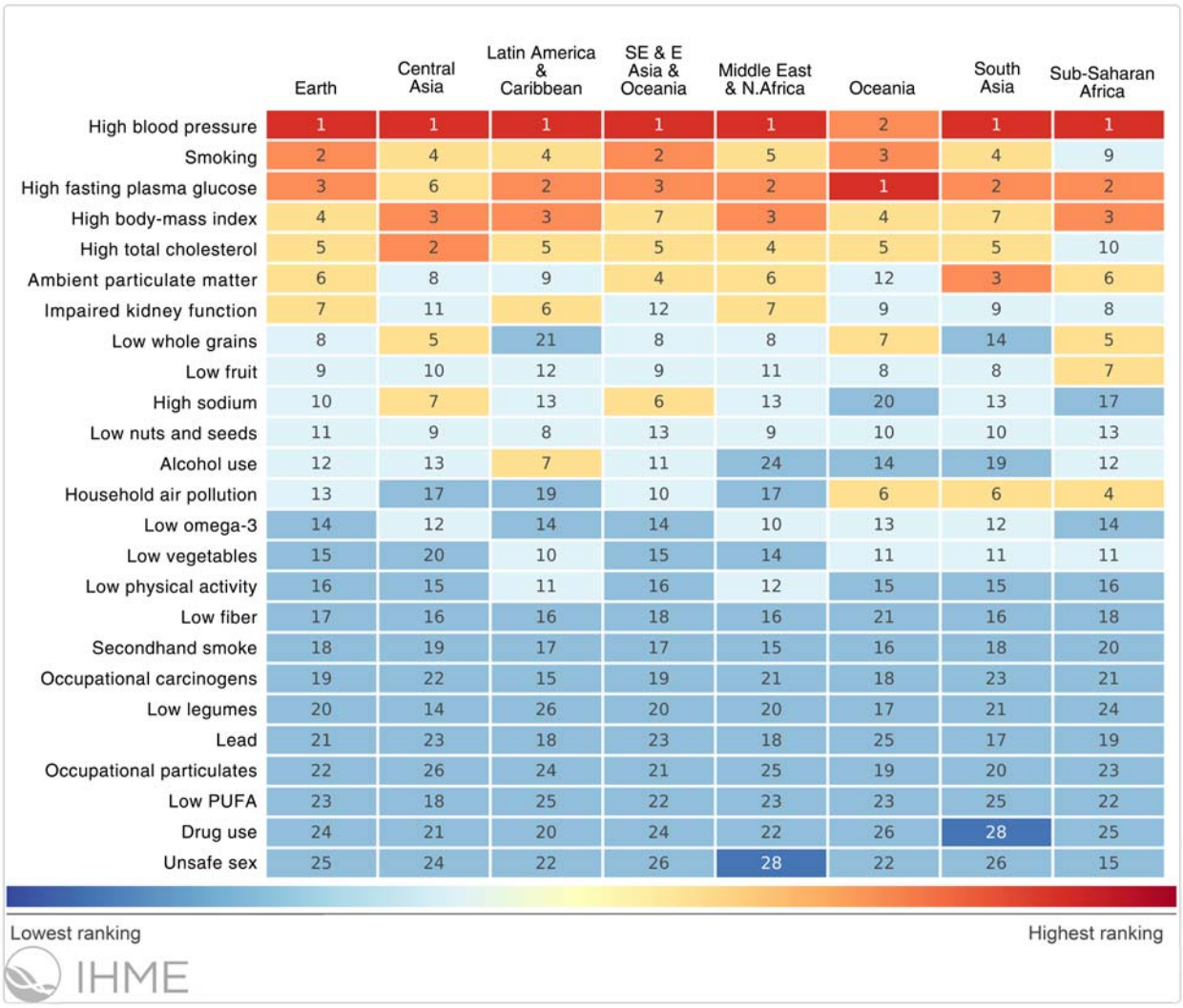

Figure 10. Deaths caused by NCDs in 2016, for both sexes, all ages, according to rank within each region. Global ranks are shown in the first column. Source: IHME 2017.

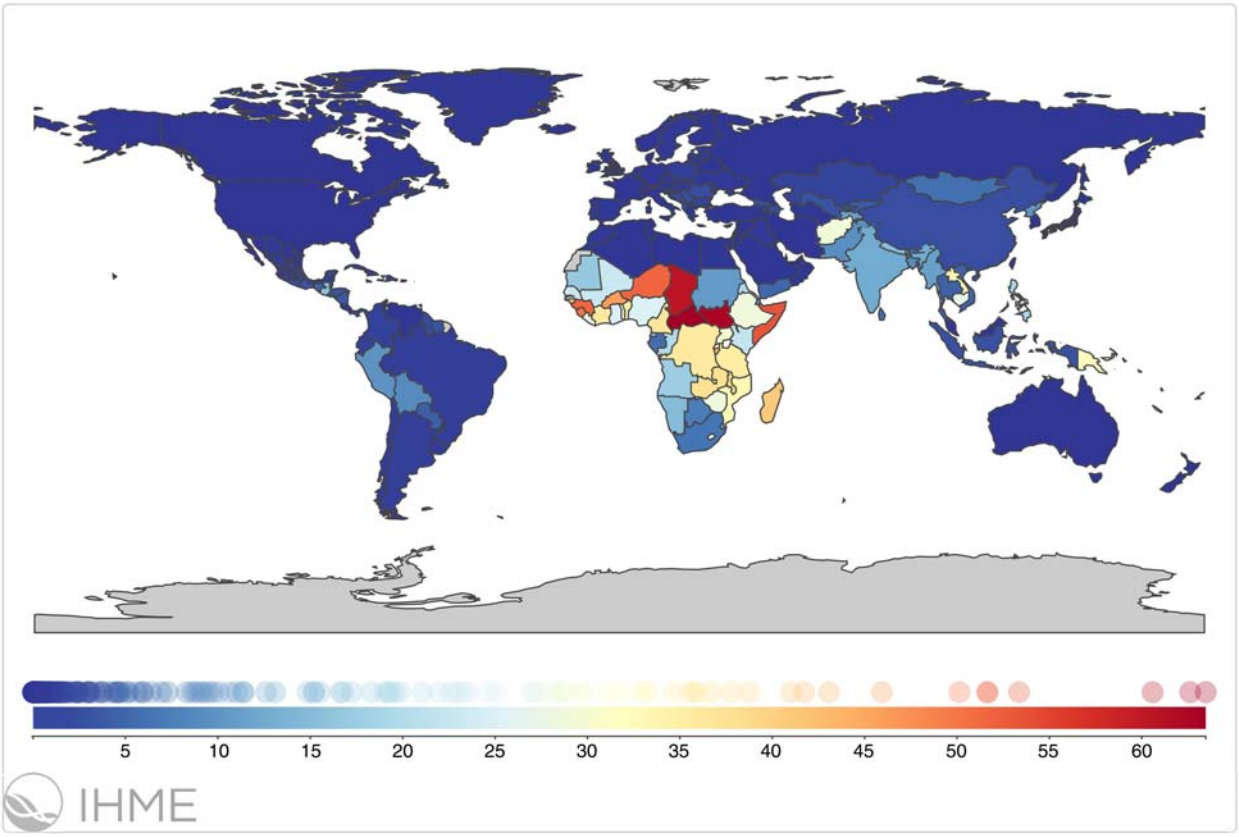

Figure 11A. LRI death rates per 100,000 population attributable to HAP from solid fuels, both sexes, all ages, in 2016. Source: IHME 2017.

respectively in sub-Saharan Africa and Central Asia over the same period (see Figure 13).

As shown in Figure 14, approximately 20% of premature deaths and DALYs from COPD worldwide have been attributed to HAP. By comparison, roughly the same percentage of COPD deaths have been attributed to ambient $PM_{2.5}$ (27% of deaths and DALYs) whereas smoking and environmental tobacco smoke (ETS) exposure together account for about 40%. Note that the percentages shown here cannot be added but can be compared. In low-sociodemographic index countries, the percentages of COPD deaths and DALYs (45% and 39% respectively) attributed to HAP are nearly double the percentage worldwide (Figure 15). These percentage contributions exceed those from smoking and ETS in these countries and more nearly approximate the worldwide toll from tobacco use.

HAP is associated with approximately 40% of the disease burden from lung cancer in countries with a low sociodemographic index. Approximately one quarter of all IHD and stroke burden has been attributed to HAP exposure in these countries, much higher than the approximately 10% of burden for each disease that is observed globally. In low-sociodemographic index countries, HAP is responsible for a higher proportion of the disease burden (from IHD, stroke, COPD, and lung cancer) than is ambient air pollution (Figure 15). The reverse is true when examining trends at the global level. This shows the disproportionate impact of HAP in low-sociodemographic index countries.

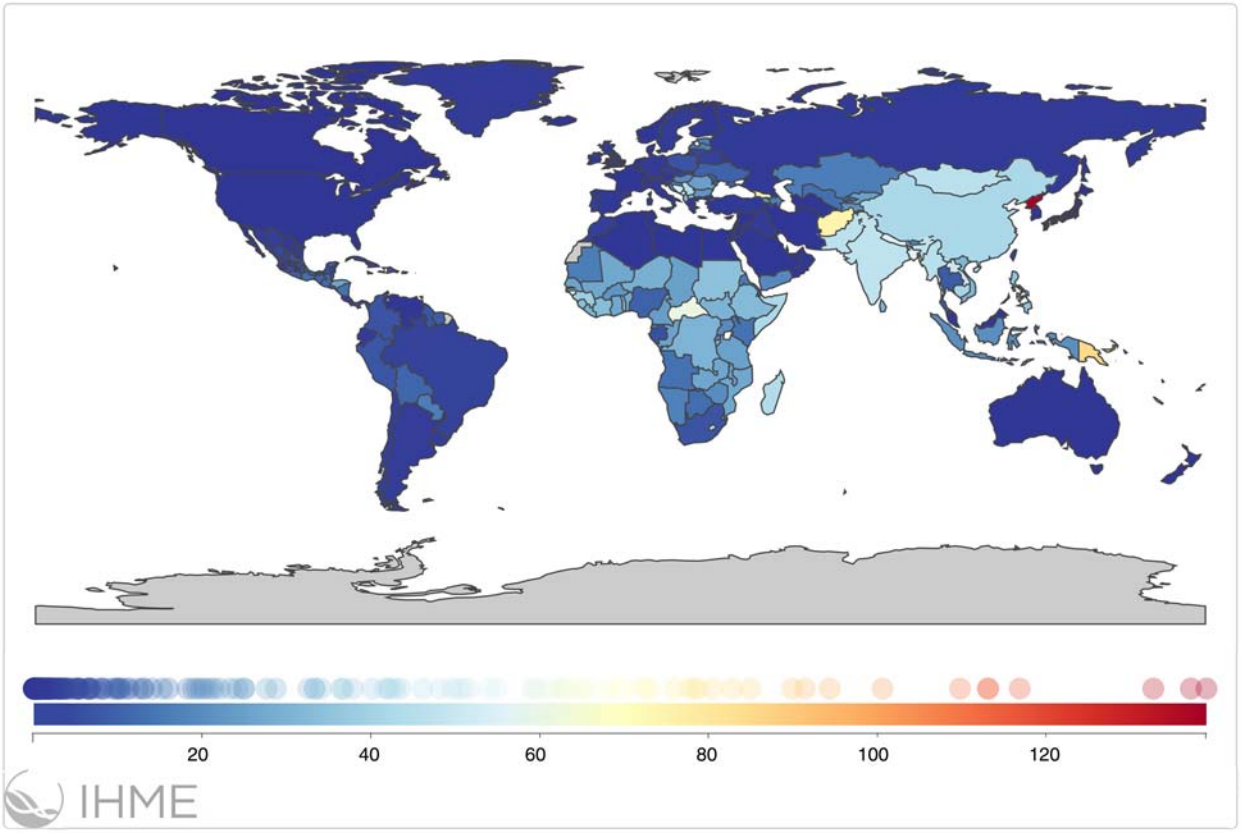

Figure 11B. NCD death rates per 100,000 population attributable to HAP from solid fuels, both sexes, all ages, in 2016. Source: IHME 2017.

Women and children accounted for between 45% and 50% of all premature deaths from HAP. For women in low- and middle-income countries, HAP was among the top causes of deaths from NCDs like stroke, COPD, lung cancer and heart disease (IHME 2017; WHO 2016). In sub-Saharan Africa, HAP exposure was the single greatest health risk identified by the WHO GBD Project (WHO 2016) and among the top ten risk factors identified by IHME (2017) for women and girls. With regard to NCDs specifically, the number of female deaths attributable to HAP in sub-Saharan Africa has risen from ~90,000 in 1990 to 123,000 in 2016. Household air pollution has fallen from the second most important risk factor for female NCD deaths in sub-Saharan Africa in 1990 (behind high systolic blood pressure) to fourth most important risk factor in 2016 (behind high blood pressure, high body mass index, and high fasting plasma glucose) (IHME 2017).

The impact of HAP on lung function is made clear by the discrepancies in lung cancer statistics among women living in China, where there is a high prevalence of solid fuel use, and the United States, where there is not; Chinese nonsmoking women have three times the lung cancer mortality compared to nonsmoking women in the United States (Gordon et al. 2014). Nonsmoking women account for 83% of all lung cancer cases in East and South Asia, compared with 15% in the United States (Gordon et al. 2014). HAP is also likely to be the most important contributor to COPD in nonsmokers (WHO 2016), though not all studies have found evidence of this association (see *Respiratory Diseases* section).

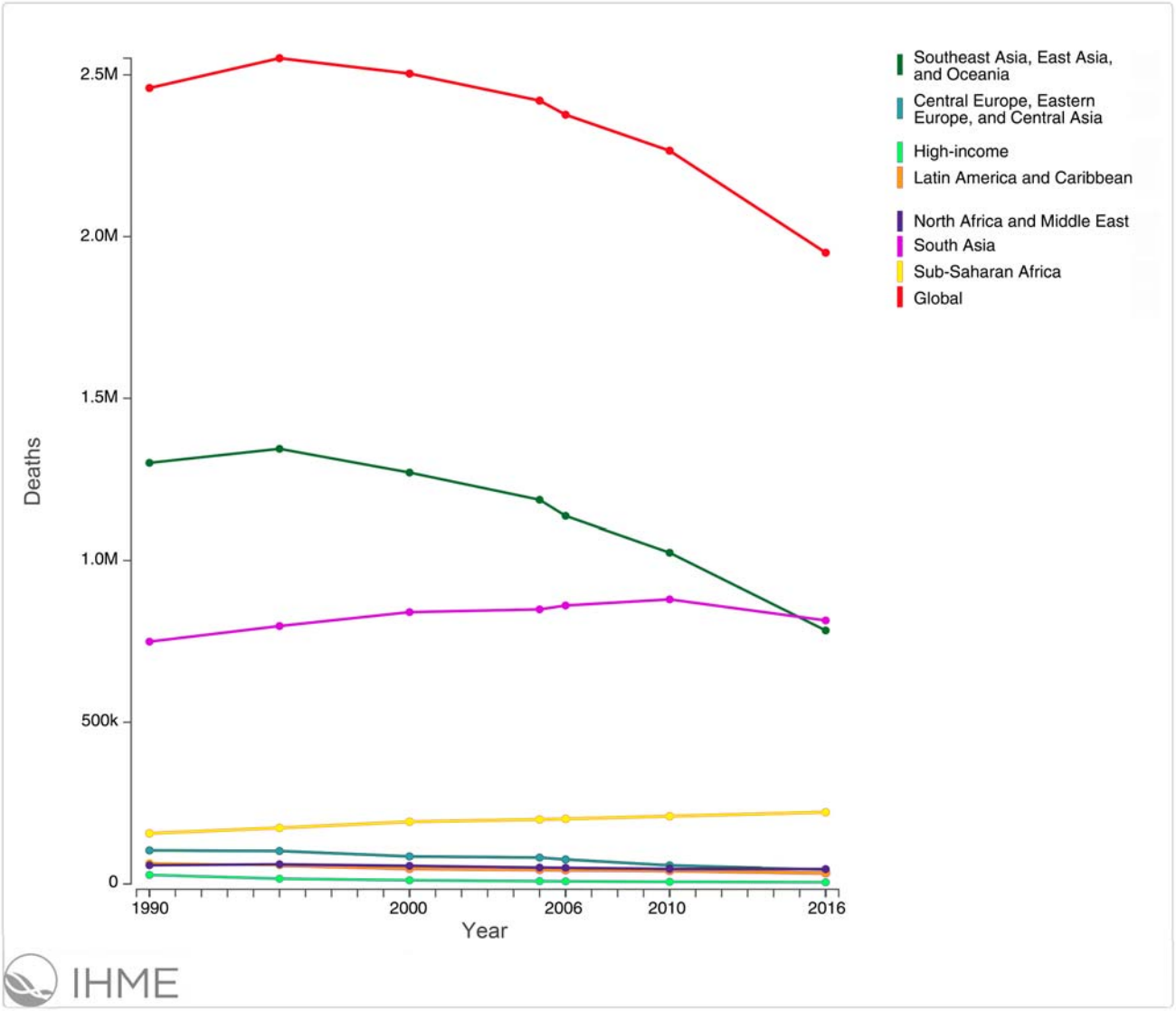

Figure 12. Trends in deaths caused by NCDs and attributable to HAP from solid fuels, 1990–2016, by IHME GBD region for both sexes, all ages. Source: IHME 2017.

The WHO has declared that a focus on HAP is an important component of the action plan for the control of NCDs in the WHO Southeast Asia Region, where NCDs are the leading cause of death (WHO 2016).

Changing Burden Estimates and Estimation Methodologies

Since 2010, GBD estimates from WHO and IHME have been revised at irregular intervals but are now updated yearly by IHME. For many disease and risks, changes in burden have been fairly straightforward and represent changes in background disease rates or an acknowledgement of new or re-emerging outcomes of interest. HAP estimates, however, have varied widely in the past decade as a result of both changes in background disease rates and changes in the methods used to estimate HAP's impact.

Between the initial estimates in 2000 and those in 2010, estimates appeared to increase substantially from 1.6 million premature deaths attributable to HAP in 2000 to 3.9 million premature deaths in 2010. The underlying

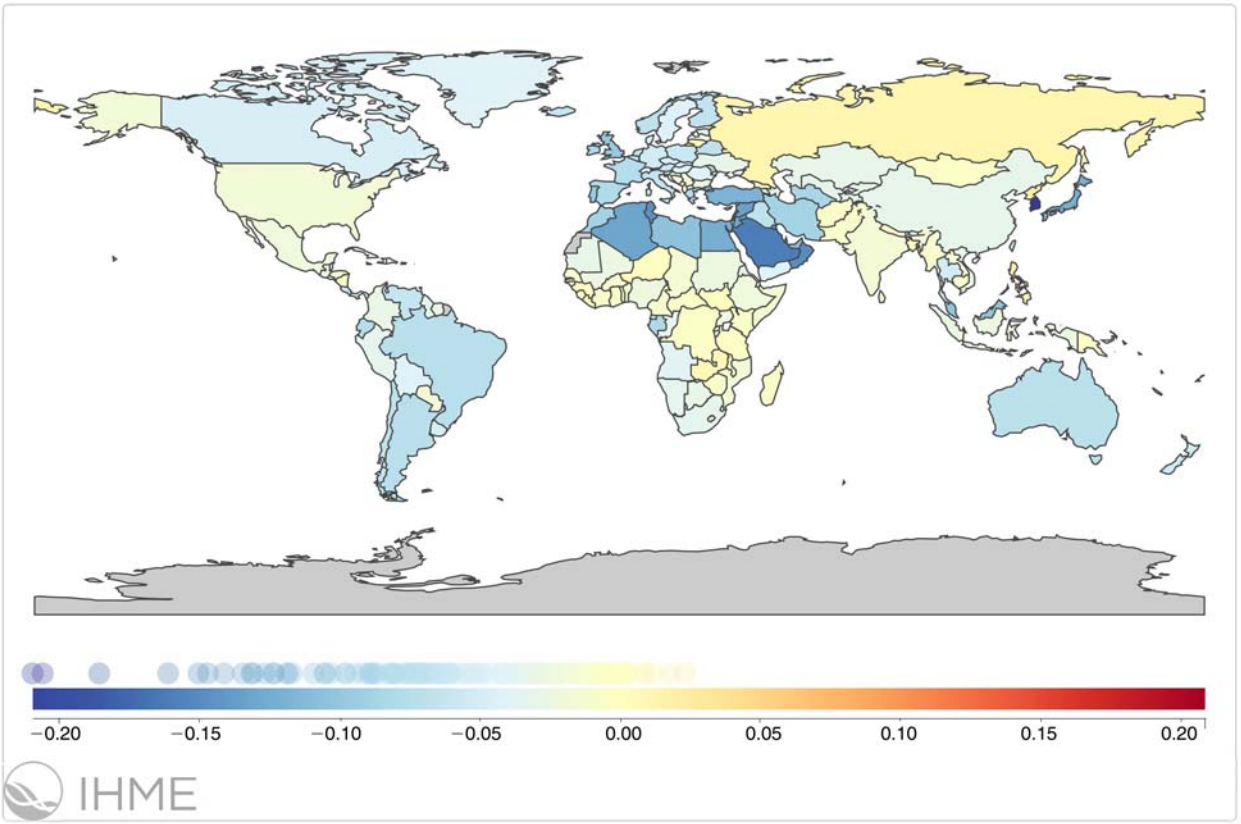

Figure 13. Annual percentage change in NCD deaths per 100,000 attributable to HAP from solid fuels, 1990–2016, for both sexes, all ages. Source: IHME 2017.

factors responsible for this shift are complex and include changes that both decreased and increased attributable burden-of-disease estimates (Smith KR et al. 2014), including:

- Vastly improved survey-based estimates of the percentage of households using solid fuels, spearheaded by WHO (Bonjour et al. 2013). These surveys indicated that 41% of households used solid fuels as a primary fuel for cooking, down from the 50% estimate used in the 2000 GBD Comparative Risk Assessment;
- COPD RRs decreased based on updated systematic reviews and meta-analyses;
- Background acute LRI rates decreased as a result of improved nutrition and vaccination;
- Improved evidence allowed (1) inclusion of new disease categories, including IHD, stroke, and cataracts, and (2) inclusion of lung cancer risks from biomass fuel use (not just coal);
- Inclusion of effects for men; and
- Inclusion of the impact of HAP via its contribution to ambient air pollution.

As a result of these changes, and due mainly to the large global disease burden of cardiovascular disease, the 2010 estimates were both significantly higher than those from 2000 and indicated a significant burden in adult populations. LRIs represented a diminished — though still substantial — burden of disease attributable to HAP.

Since 2010, IHME estimates have been revised in 2015 for the year 2013, in 2016 for years 2015 and 2016, and are undergoing revision in 2018 for 2017. Comparing current figures with previous ones is not advisable; IHME revisions supersede all previous estimates and previous years' burdens are re-estimated using newer methodologies. Previous estimates using methodologies current at the time are not readily available via IHME's website. In 2013, HAP was estimated to account for 2.9 million premature deaths globally each year (95% CI = 2.5–3.3). In 2015, the estimate was 2.8 million (2.2–3.6) and in 2016, 2.6 million (2.2–3.0). These changes have been attributed to changes in (1) underlying

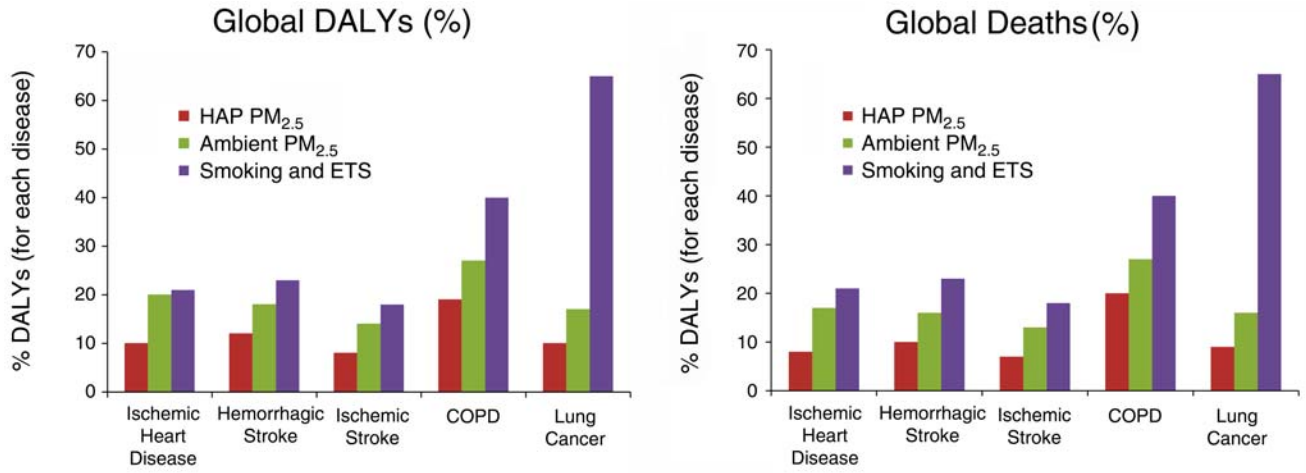

Figure 14. Comparison of deaths and DALYs attributable to HAP vs. other risk factors globally in 2016. Note: These percentages cannot be added since there is some overlap. They are for comparison purposes only. ETS = environmental tobacco smoke. Source: IHME 2017.

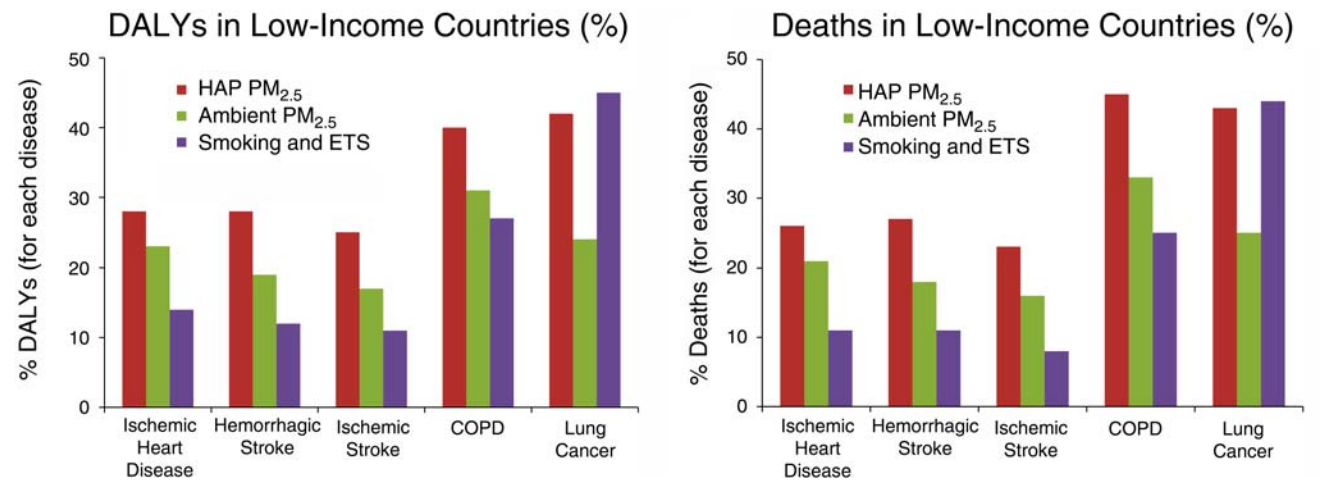

Figure 15. Comparison of deaths and DALYs attributable to HAP vs. other risk factors in low-income countries in 2016. Note: These percentages cannot be added since there is some overlap. They are for comparison purposes only. Countries included are in the low SDI category. ETS = environmental tobacco smoke; SDI = sociodemographic index. Source: IHME 2017.

disease burden, (2) the IER curves, and (3) the methods used to estimate exposure to HAP with greater spatial resolution. They also reflect declines in exposure to HAP over time in many regions of the world (Figure 2).

In addition to IHME estimates, WHO released a separate estimate of the impact of HAP for 2012 (Prüss-Üstün et al. 2016) that estimated approximately 4.3 million attributable premature deaths. WHO's estimate varies from IHME's in the following ways: (1) since 2013, IHME has utilized regional or national exposure estimates; WHO uses global estimates based on those used in GBD 2010, which apply modeled estimates from India to all households globally (Smith KR et al. 2014); (2) WHO includes kerosene-related impacts; (3) IHME and WHO use different background disease estimates.

Such variations in the estimates can prove difficult to explain to policy makers, nongovernmental organizations, and nonacademic health practitioners. However, they are explained by different analytical choices reflecting uncertainties in the science; all of the estimates support the conclusion that the potential impacts of HAP on human health are large and substantial.

Figure 16. HAP often contributes to ambient air pollution, exposing people to health-harmful pollution on broad geographical scales. Photo: Ajay Pillarisetti, by permission

CONTRIBUTION OF HOUSEHOLD AIR POLLUTION TO AMBIENT AIR POLLUTION

Household air pollution is now understood to have health effects beyond the houses in which the air pollution is emitted (Figure 16). Cooking and heating emissions are a significant source of outdoor (or ambient) particulate air pollution ($PM_{2.5}$) in many regions of the world and must be addressed in strategies to improve air quality (Chafe et al. 2014; Hill et al. 2017).

Estimates of the global health burden caused by population-wide exposure to outdoor air pollution ($PM_{2.5}$) from household cooking and heating with solid fuels range from 308,000 (for all residential sources; Butt et al. 2016) to 370,000 (for cooking alone; Chafe et al. 2014) (Table 7). Another paper that examined sectoral contributions to total outdoor air pollution ($PM_{2.5}$ and ozone) estimated that residential energy emissions contribute approximately 31% of the air pollution. It was unclear what was

Table 7. Global Estimates of Burden of Disease from Exposure to Outdoor Air Pollution Caused by Household Combustion of Solid Fuels

Author / Year	Burden of Disease Estimate	Region	Emissions Considered	Other Notes
Butt et al. 2015	308,000 premature deaths	Global	All residential	Adult (>30 yrs) only
Chafe et al. 2014	370,000 premature deaths 9.9 million DALYs	Global	Cooking	Year: 2010
Chafe et al. 2015	110,000 premature deaths 2.2 million DALYs	Global	Heating	Year: 2010

considered within the "residential energy" source category (Lelieveld et al. 2015).The total toll from exposure to air pollution globally was 3.3 million premature deaths in 2010.

In sub-Saharan Africa, the overall proportion of annual average ambient $PM_{2.5}$ attributable to household cooking is high (<37% in 2010). In East Asia and South Asia, while the proportion of $PM_{2.5}$ from household sources is relatively less (10% and 26% respectively) due to the presence of other significant emissions sources, the concentration of $PM_{2.5}$ that comes from household sources tends to be higher (Chafe et al. 2014). In 2010, household cooking emissions accounted for an estimated 7.3 $\mu g/m^3$ annual population-weighted average $PM_{2.5}$ in East Asia, and 8.6 $\mu g/m^3$ $PM_{2.5}$ in South Asia, whereas the estimate was 1.2–2.8 $\mu g/m^3$ in sub-Saharan African regions in the same year.

In a burden-of-disease study of major air pollution sources in China in 2013, the Health Effects Institute found that household solid fuel combustion was the second largest sector (behind industry) in contribution to annual average ambient $PM_{2.5}$-attributable mortality (GBD MAPS Working Group 2016). The health toll from household heating and cooking with biomass and coal in China was estimated to be 177,000 deaths (19% of the mortality attributable to ambient $PM_{2.5}$ in 2013) larger than that of industrial coal (155,000 deaths), transportation (137,000 deaths), or coal combustion in power plants (86,500 deaths). Ambient $PM_{2.5}$ deaths attributable to household biomass combustion alone (136,000) were roughly equivalent to deaths attributable to industrial coal and transportation.

A related study in India found household biomass burning to be the largest contributor to ambient $PM_{2.5}$ and the related health burden (GBD MAPS Working Group 2018). Household biomass burning was responsible for 267,700 deaths or nearly 25% of the deaths attributable to $PM_{2.5}$, making it the most important single anthropogenic source related to mortality in 2015. These burden estimates did not include the additional substantial burden from indoor exposure to biomass burning in the home. By comparison, coal combustion, roughly evenly split between industrial sources and thermal power plants, was responsible for 169,300 deaths (15.5%) in 2015. The open burning of agricultural residue was responsible for 66,200 $PM_{2.5}$-attributable deaths (6.1%).

These studies of the contribution of specific sources to disease burden rely on estimates of annual average exposures over extended time frames. Shorter term, seasonal contributions of household solid fuel combustion to ambient air pollution can be higher. Further work is necessary to corroborate such findings in other settings and to understand their implications for health.

POTENTIAL FUTURE CHANGES IN THE HOUSEHOLD AIR POLLUTION BURDEN OF DISEASE

From 1990 to 2008, nearly two billion people gained access to electricity, more than the corresponding population increase of 1.4 billion people over that period (Global Energy Assessment 2012). The total proportion of households using solid fuels is decreasing continuously, from 62% in 1980, to 53% in 1990, to 46% in 2005, to 41% in 2010 (Bonjour et al. 2013), and to about 31% in the latest GBD 2016 results (IHME 2017). However, the absolute number of people remains substantial — nearly a third of the global population.

Demographic transitions, such as growing and aging populations, also affect vulnerability to the health effects of HAP. Figure 17 shows that many countries experienced declines in HAP exposure (see yellow bars) in 2013 relative to 1990. At the same time, they experienced decreases in age-standardized mortality rates (see gray bars) for reasons that are likely independent of and unrelated to HAP exposure. The decreases are offset by population growth (see blue bars) and population aging (see orange bars). The result is a net increase in HAP-attributable mortality in most major countries, including India, where the potential health benefits of small reductions in exposure were counteracted by growth and aging of the population, which led to increases in NCD deaths in adults. Brazil and China had net decreases in HAP-related deaths over the same time period.

The International Energy Agency (IEA) reported that regional demographic trends, rising energy use, and urbanization, especially in Asia, mean that the number of premature deaths attributable to ambient air pollution will continue to grow, from approximately 3 million in 2016 to 4.5 million in 2040 (IEA 2016). Global population-weighted $PM_{2.5}$ increased by 11% between 1990 and 2015, with its most rapid increase between 2010 and 2015 (Cohen et al. 2017). Notably, exposures increased between 2010 and 2015 in Bangladesh and India and remained high in China and Pakistan (Cohen et al. 2017).

In China, an aging population is becoming more vulnerable to the effects of air pollution on human health, even though aggregate pollutant emissions are in decline (IEA 2016). HEI's reports on China and India underscore this dynamic, finding that China's and India's aging populations will likely become more susceptible to the diseases most closely linked to air pollution (GBD MAPS Working Group 2016, 2018). Globally, trends in $PM_{2.5}$-attributable mortality among countries generally reflect changes in $PM_{2.5}$-attributable mortality from cardiovascular disease (Cohen et al. 2017). However, it is notable that exposure to particulate air pollution contributes to deaths among children as well the elderly: it contributed to 202,000 child deaths from LRI in 2015 and 17.4 million DALYs (Cohen et al. 2017).

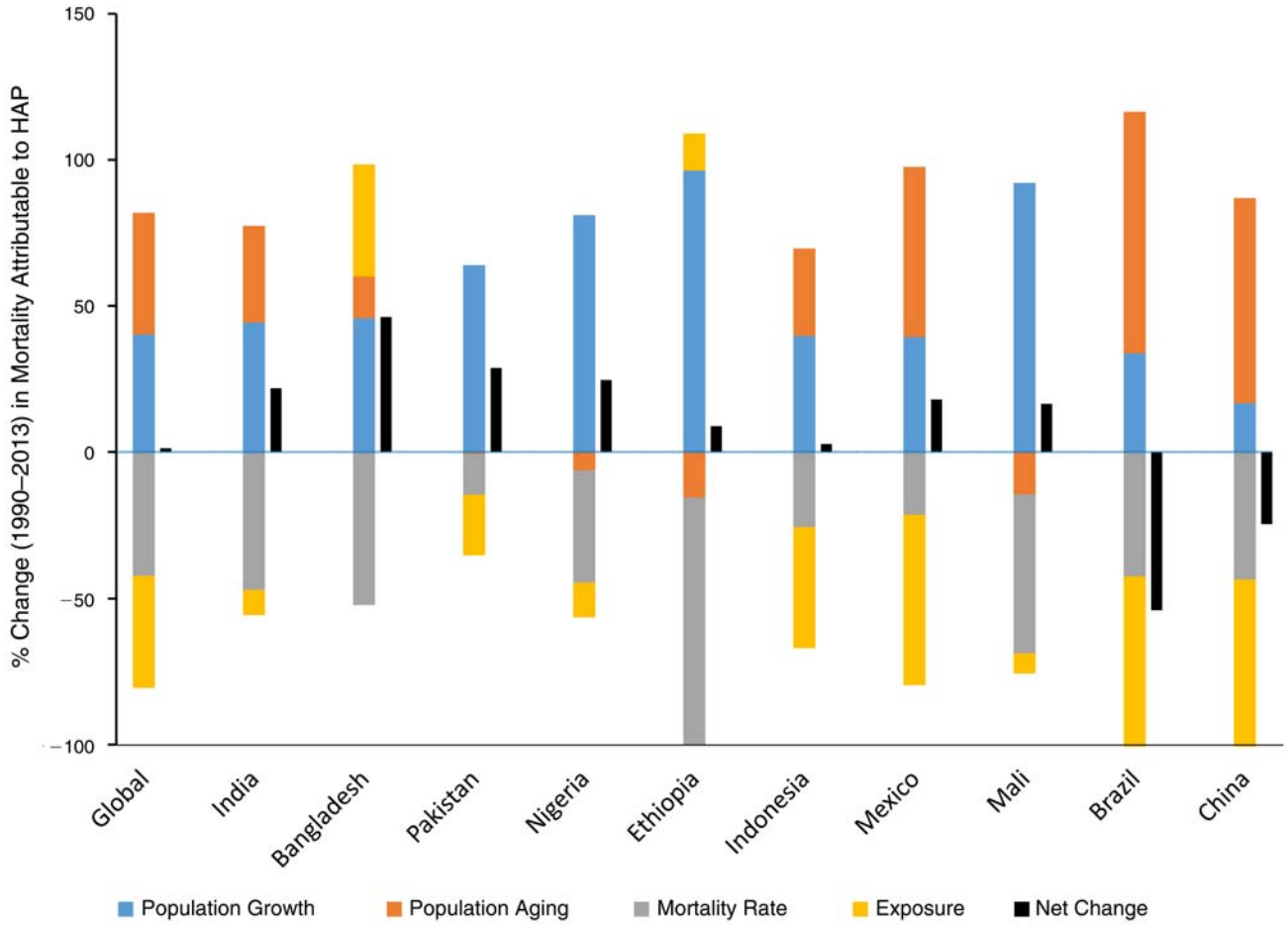

Figure 17. Drivers of trends in mortality attributable to HAP globally and in 10 countries. Source: Aaron Cohen, by permission.

Transitions away from polluting fuels and inefficient stoves to cleaner fuels and more efficient stoves are happening at different rates across rural and urban areas within countries, between countries, and between regions. For example, the proportion of people using solid fuel across China has decreased from 64% in 1990 to 46% in 2010; but in rural China, about two-thirds of people still use solid fuels, particularly coal, as their main source of energy for cooking and heating (Gordon et al. 2014). Migration to cities also can change fuel use patterns and consequently HAP emissions, sometimes decreasing estimated exposure to HAP (Aunan and Wang 2014). One study estimated that annual mean exposure to $PM_{2.5}$ among migrants from rural to urban areas in China dropped by 215 $\mu g/m^3$ (Aunan and Wang 2014).

In China, two types of scenarios for controlling emissions and exposures were evaluated for the year 2030 in the GBD MAPS project (GBD MAPS Working Group 2016). The *business-as-usual* scenario relies more on existing energy uses but also assumes gradual penetration of low-sulfur coal, replacement with advanced coal stoves and advanced biomass stoves (e.g., better combustion conditions or catalytic stoves), as well as transition to use of clean fuels in both urban and rural areas. A second *policy-control* scenario assumes much higher rates of adoption of new technologies and fuels than in the business-as-usual scenario. The more significant shifts to cleaner energy envisioned by the policy-control scenario are projected to decrease the expected disease burden in 2030 by about 63%, or over 89,000 deaths, and nearly 1.4 million DALYs (Table 8) compared with the business-as-usual scenario.

Table 8. Reductions in Health Impacts Attributable to Residential Burning of Biomass and Coal in China by Scenario and Year

	2013	2030 BAU	2030 Alternative PC	Burden Reduction PC−BAU (%)
Deaths	177,490	142,540	52,850	−89,690 (63)
DALYs	3,563,502	2,353,050	888,250	−1,464,800 (62)

BAU = business-as-usual scenario; PC = policy-control scenario.

Table 9. Reductions in Health Impacts Attributable to Residential Burning of Biomass in India by Scenario and Year

	2015 Baseline	2050 REF	2050 S2	2050 S3	Burden Reduction S2−REF (%)	Burden Reduction S3−REF (%)
Deaths	267,700	526,400	366,800	19,300	−159,600 (30)	−507,100 (96)
DALYs	7,373,200	10,696,300	7,450,000	391,400	−3,246,300 (30)	−10,304,900 (96)

REF = reference scenario; S2 = ambitious scenario; S3 = aspirational scenario.

In India, in addition to a reference, or business-as-usual scenario, two scenarios were designed to reflect different strategies for reducing emissions from major sources; they differ with regard to the contributions of different energy sources and their prioritization and aggressiveness of source-specific emission reductions. They were projected to the year 2050, under assumptions that a longer time was needed to implement the new policies in India. For domestic biomass burning, the scenarios reflect different percentages of households converted to clean energy use, with the most aspirational scenario (S3) involving nearly complete elimination of the use of traditional solid fuels in favor of LPG, electricity, and gasifiers, resulting in an over 95% reduction in mortality (>500,000 deaths) (Table 9).

It is possible that, over a relatively short period (~10 yr), the region with the highest *avoidable* burden of disease from HAP could become sub-Saharan Africa, rather than South or Southeast Asia for two main reasons: (1) transitions to liquid fuels are happening faster in South Asia and Southeast Asia than in other regions, such as sub-Saharan Africa, and (2) sub-Saharan Africa still has a high overall burden of disease from LRI, which currently accounts for a greater burden of DALYs than do cardiovascular disease, COPD, and lung cancer combined (Kuhn et al. 2016).

ECONOMIC BURDEN OF HOUSEHOLD AIR POLLUTION

According to the best available estimates, HAP exposures lead to an annual global welfare loss of about $1.5 trillion (World Bank and IHME 2016). Insight into the economic burden from HAP-related health effects is important for two interrelated reasons. First, reliable estimates of the economic costs of HAP may help spur governments, multilateral lenders, and private foundations to invest in HAP reduction efforts. Second, estimates of the economic burden of HAP exposure are a critical input into a cost–benefit analysis, which can provide an analytic answer to the question of *how much* HAP investment reduction is most efficient. Recent efforts to assess the economic burden of HAP exposure — and its corollary, the economic benefits of exposure reduction — fall into two broad categories.

First, in 2016 the World Bank and IHME monetized the burden-of-disease estimates described above (World Bank and IHME 2016). Results for both HAP and ambient $PM_{2.5}$ are summarized in Figure 18. The World Bank–IHME effort focused on mortality, given that regulatory impact analyses in the United States have consistently found that avoided mortality dominates the economic benefits of air pollution reduction. Their approach relies on two distinct methods for estimating the economic burden of air pollution exposure.

- *A welfare-based approach that builds on a standard estimate of willingness-to-pay studies that estimate the value of a statistical life (VSL).* The report uses a base VSL of US$ 3.83 million, which is adjusted to account for country-specific factors that have been shown in other studies to affect willingness-to-pay for mortality reduction. Income is the most salient of these local adjustment factors. The resulting welfare loss number ($1.5 trillion per year) is analogous to benefits estimates typically used by the U.S. Environmental Protection Agency and other high-income governments in the context of cost–benefit analysis.
- *A lost labor approach that estimates the expected loss of income experienced by those who die prematurely from HAP exposure.* This measure is primarily of interest because it lends itself to inclusion in a given country's system of national accounts (e.g., as a component in adjusted net savings). The lost labor approach excludes several categories of economic burden, including medical costs and individuals' willingness to pay to avoid suffering.

The results of these analyses are striking. As noted above, the annual global welfare loss is approximately $1.5 trillion (for 2013, in 2011 dollars). For the lost labor approach, the net benefits are smaller but still very substantial ($94 billion for 2013 in 2011 dollars).

The counterfactual exposure (the exposure level below which health impacts are no longer calculated) that underpins the GBD analysis is extremely stringent and would require that entire populations switch to LPG, electricity, and other clean, but expensive, cooking technologies. To date, no HAP intervention has achieved levels even close to the GBD theoretical minimum exposure levels, or even the higher WHO Air Quality Guideline for $PM_{2.5}$ (Pope et al. 2017). On the other hand, the focus on mortality excludes important categories of benefits, including morbidity, climate impacts, deforestation, and reduced time gathering firewood. At present, no study has attempted to monetize the full set of benefits that flow from clean cooking (though at least two studies have estimated the joint health and climate benefits without monetizing them (Anenberg et al. 2017; Grieshop et al. 2011). (See Appendix B for more discussion on the climate benefits of HAP reductions.)

Finally, the World Bank–IHME (2016) analysis does not consider the costs that would have to be incurred to achieve the exposure reductions assumed in the GBD estimates. The only previous study to consider the global costs and benefits of clean energy transitions examined the costs and benefits of transitioning half of the HAP-exposed population to either LPG or improved cookstoves and found large net benefits (Hutton et al. 2007). For the LPG scenario, the authors found benefits (in the form of avoided

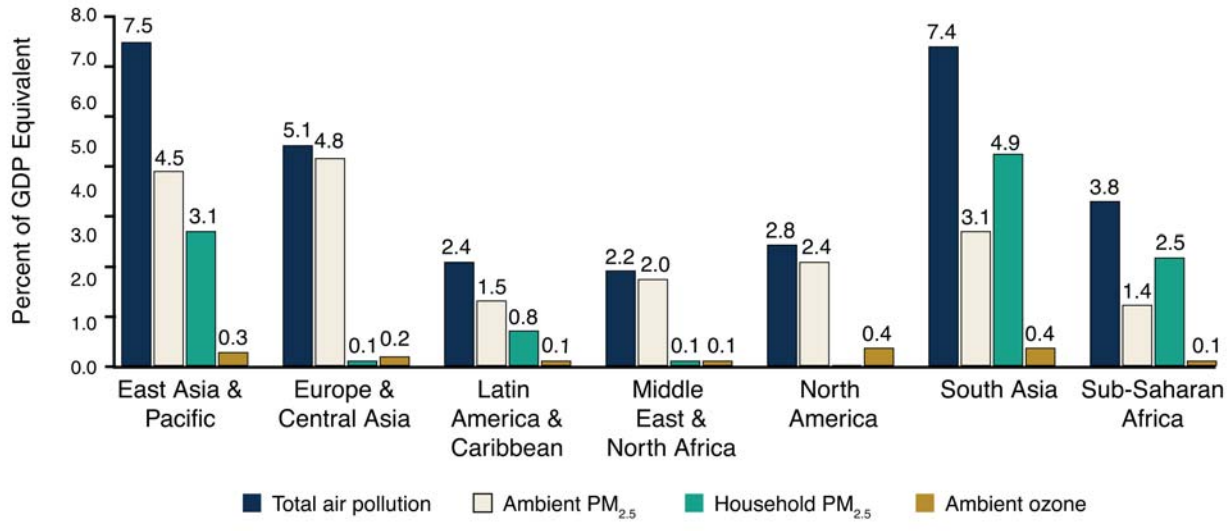

Figure 18. Welfare losses from air pollution exposure. Total air pollution damages include ambient $PM_{2.5}$, household $PM_{2.5}$, and ozone. GDP = gross domestic product. Source: World Bank and IHME 2016. License: Creative Commons Attribution CC BY 3.0 IGO.

morbidity and mortality) of $101 billion and costs of $24 billion. This prior analysis drew on estimates from 2002 and considered the following benefit categories: avoided mortality; avoided cost of health care; productivity gains; time savings; and reduced deforestation and greenhouse gas emissions.

In addition to these global assessments, several researchers have carried out what might be termed *micro-economic* benefit assessments, which explore benefits of clean cooking in specific populations. These approaches often use measured (as opposed to modeled) data to derive at least some parameters. For example, Malla and colleagues (2011) used detailed field measurements from sites in Sudan, Nepal, and Kenya to estimate intervention costs, reductions in CO exposures, fuel savings, and cooking time savings. They found that benefits substantially exceeded costs in all locations. Jeuland and Pattanayak (2012) explicitly considered uncertainty in benefits and costs through a Monte Carlo simulation analysis (which repeatedly draws values of uncertain parameters from a probability distribution, and then averages the results). Their analysis suggests that net benefits experienced by households will sometimes be highly negative. Other recent papers have carried out cost–benefit analyses for Nigeria (Isihak et al. 2012), rural China (Aunan et al. 2013), rural India (Patel et al. 2016), and in Indonesia, the Philippines, and Timor-Leste (Arcenas et al. 2010). All four of these analyses found significant net benefits from switching to cleaner cooking, but they relied on idiosyncratic methods. Toman and Bluffstone (2017) reviewed the methodological difficulty — and importance — of local estimates of the costs and benefits of clean cooking.

HEALTH BENEFITS OF REDUCED HOUSEHOLD AIR POLLUTION EXPOSURES

Many types of interventions have been implemented around the world, with varying approaches and degrees of success. Potential interventions include provision of clean fuels, improved solid fuel stoves, enhanced ventilation (e.g., use of chimney), and behavioral changes (e.g., cooking outdoors, opening the kitchen door and windows while cooking, avoiding leaning over the fire while cooking, and keeping children away from the cooking area). Clean fuels include electricity, LPG or natural gas, ethanol, biogas, and solar cookers. Improved solid fuel stoves may include natural-draft stoves and forced-draft stoves (employing a fan to increase combustion efficiency) using either unprocessed fuels (e.g., collected wood, agricultural waste) or processed fuels (e.g., biomass or charcoal pellets). The performance of these intervention types — and the individual models within each category — for reducing fuel use, HAP exposures, climate warming emissions, and other priorities varies greatly.

Many stove models are not tested for performance prior to using them in interventions, which can often lead to investments in technologies that are insufficient to achieve the project's objectives. Laboratory testing can provide an indication as to the inherent performance of the technology under ideal conditions. However, laboratory testing is typically not reflective of actual performance of the technology in the field (Johnson et al. 2008, 2011; Roden et al. 2009). This type of mismatch between laboratory and field testing is not unique to HAP research — similar disparities have been observed for vehicle emissions and many other issues. What makes the mismatch for HAP more challenging is that the problem is very decentralized (i.e., many small-scale disaggregated stove manufacturers and dispersed household-level consumers), and it occurs mainly in low- and middle-income settings where access to and affordability of cleaner fuels and technologies are limited. Thus, while the gap between the laboratory and the field can be minimized with strong national policies and enforcement for vehicle emissions, it is more challenging to address for HAP.

Estimating or measuring health improvements from cookstove interventions thus requires an understanding of the effect of the intervention on actual exposure levels in the field. Once exposure levels are known, exposure–response curves can be used to model the number of deaths and DALYs that would theoretically be averted with the measured exposure reductions. Measuring health benefits of reduced HAP in the field is more complex because changes in other health determinants and population dynamics can obscure the relationship between the intervention and health outcomes. This section summarizes the state of the science regarding field exposure measurement, methods, and tools available for modeling health benefits from exposure reductions and literature estimates of the theoretical health benefits that would come from HAP exposure reductions. It also reviews recent evidence from field intervention studies, including ongoing randomized control trials, as well as challenges in terms of demonstrating health benefits from field interventions in practice.

MEASURING HOUSEHOLD AIR POLLUTION: RECENT ADVANCES AND CONTINUED CHALLENGES

Estimating the impact of HAP, and of the interventions undertaken to reduce it, requires accurate exposure assessment. Combustion of solid fuels in simple stoves releases hundreds of compounds into the household and near-household environment, many of which are known human

toxins and carcinogens (Naeher et al. 2007). This release, however, does not necessarily result in high exposure for an individual. To begin, we clarify the distinction between (1) emissions, (2) concentrations, (3) exposures, and (4) dose in the context of HAP (adapted from Pillarisetti et al. 2016; Smith and Pillarisetti 2017; WHO 2014b).

- *Emissions* refer to the rate of release of a pollutant per unit of time (e.g., mg pollutant per second) or per unit of fuel (e.g., mg pollutant per kg wood burned). Emissions are often measured directly from the plume of smoke arising from combustion sources. Emissions measurements can also be taken in a laboratory — where stoves are fed uniform fuel in a uniform fashion. Those measurements tend to underestimate conditions in households, where behavioral and fuel-related heterogeneity are common (Johnson et al. 2008, 2011; Roden et al. 2009).
- *Concentrations*, measured in terms of mass of pollutant per volume of air (e.g., microgram pollutant per cubic meter air), are at minimum a function of (a) emissions, (b) the characteristics of the microenvironment in which combustion occurs, and (c) deposition and exfiltration. For HAP, concentrations are often measured in kitchens or other living areas (Balakrishnan et al. 2011; Northcross et al. 2015) and are referred to as *area measurements*. Area measurements do not necessarily take into account whether people are actually present — for instance, a monitor measuring $PM_{2.5}$ on the wall of a kitchen for 24 hours may not be representative of a cook's exposure to $PM_{2.5}$ as he or she moves in and out of the kitchen, into other rooms, or outdoors.
- *Exposure* (or personal exposure) describes the spatio-temporal relationship between individuals and polluting sources in their environs. An individual's daily average exposure depends on the number, type, and duration of contact with polluting sources. For instance, a cook may be exposed to smoke from her own open fire, from outdoor burning when working, and from her neighbor's fire. Exposures can be assessed by (a) having a participant wear a monitor that follows him or her in time and space; (b) by measuring pollutant concentrations and the time spent in specific microenvironments, and then calculating a time-weighted average; and/or (c) by measuring biomarkers of exposure.
- *Dose* refers to the internalized, biologically relevant component of, in this case, an inhaled pollutant. Assessing doses of combustion-related particles is difficult; dose can be estimated by deriving ventilation rates (through, for example, accelerometers, which measure movement, coupled with personal-exposure monitors) and relating measured concentrations with those ventilation rates. Additionally, biomarkers of exposure can also be coupled with environmental measurements to estimate dose, though much work is needed on identifying the proper suite of both environmental and biomarker-based species to measure.

Emissions can be measured in both the laboratory and the field, though discrepancies frequently exist between the same stove measured in controlled laboratory and then in real-world conditions. In 2014, the WHO published emission rate targets for household fuel combustion (WHO 2014b) (Table 10) generated through repeated simulation of an indoor kitchen using a single compartment box model (Johnson et al. 2011). These targets were judged to be required to meet WHO annual average Air Quality Guidelines and Interim Target-1 for $PM_{2.5}$ (35 $\mu g/m^3$) and the 24-hour average air quality guideline for CO, using assumed values for kitchen volume, air exchange rate, and duration of device use per 24 hours. Even with stoves that meet these emission rate targets, however, actual

Table 10. WHO Targets for Emission Rates from Household Fuel Combustion

		Emission Rate Targets	
Recommendation	Pollutant	Vented	Unvented
Emission rates from household fuel combustion should not exceed the following emission rate targets for $PM_{2.5}$ and CO	$PM_{2.5}$	0.80 (mg/min)	0.23 (mg/min)
	CO	0.59 (g/min)	0.16 (g/min)

Source: WHO 2014b.

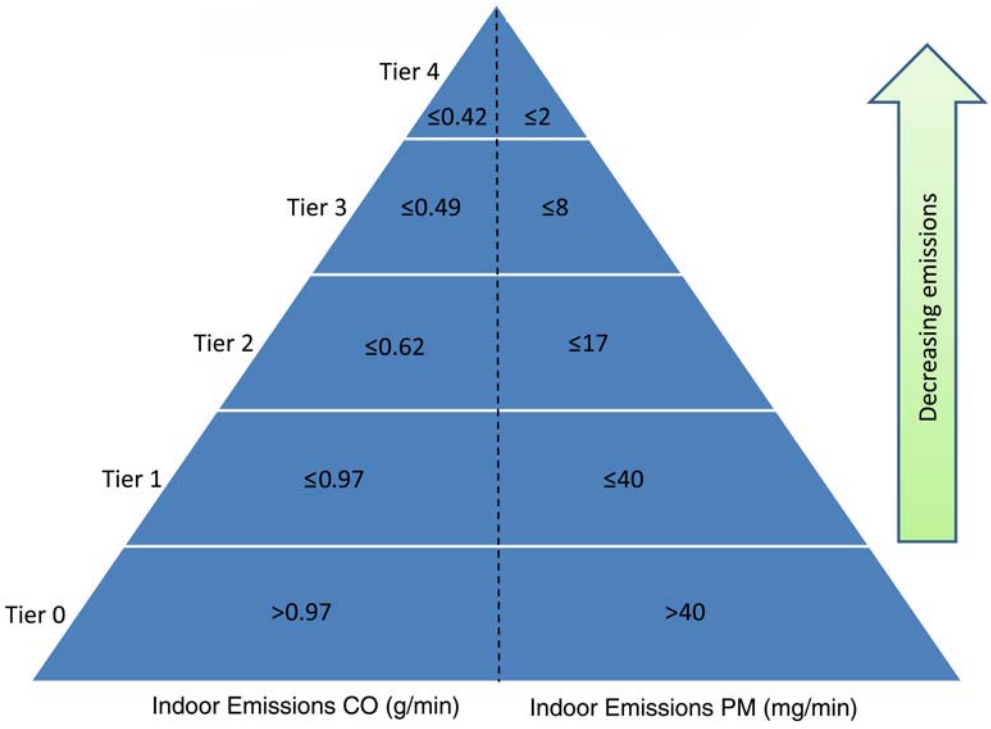

Figure 19. Tiers of performance from the International Workshop Agreement for indoor emissions of CO and PM. Data source: International Organization for Standardization 2012.

exposures may not be reduced to the same degree due to stove and fuel stacking, heterogeneity in fuel types and conditions, and continued exposure to neighborhood and ambient pollution. For example, the WHO used a simple model to estimate that gas stoves would meet Interim Target-1 for $PM_{2.5}$ in 99% of homes, but studies show that in practice such stoves led to concentrations in homes around 70 $\mu g/m^3$. Field exposure measurements are thus necessary to judge the effectiveness of interventions at reducing actual exposures.

Different stove technologies are commonly evaluated according to tiers of performance defined in 2012 by the International Workshop Agreement, developed as part of an International Organization for Standardization consensus process that may be a first step towards formal standards for cookstoves (International Organization for Standardization 2012). The International Workshop Agreement defined separate indicators of cookstove performance for efficiency, total emissions, indoor emissions, and safety. As shown in Figure 19, each of these indicators has five associated tiers, from Tier 0 to Tier 4, with Tier 4 indicating the best performance. Many cookstoves currently on the market are tested according to this methodology and assigned a tier for each indicator tested. This system is a simple and useful way to communicate performance but does not necessarily reflect actual performance of the stove at reducing exposures in field settings.

Therefore, while emissions are often measured in HAP field studies, for the purposes of this review we focus on HAP personal-exposure measurements. Measuring area concentrations alone tends to misestimate exposure; that is, the relationship between personal exposures and area concentrations varies by study site, stove type, kitchen configuration, behavior, and other factors (Clark et al. 2013; Northcross et al. 2015).

Household Air Pollution Measurements

The most commonly measured pollutants in HAP studies are CO and $PM_{2.5}$ (Balakrishnan et al. 2011; Clark et al. 2013; Northcross et al. 2015; Quansah et al. 2017), though there is an increasing focus on: (a) measuring environmental concentrations of and exposures to polycyclic aromatic hydrocarbons (Chen et al. 2017; Downward et al. 2014), volatile organic compounds (Wangchuk et al. 2015), and

black carbon (Baumgartner et al. 2014; Norris et al. 2016; Van Vliet et al. 2013); and (b) identifying and quantifying biomarkers of exposure and of early and chronic effect (Alexander et al. 2017; Caravedo et al. 2016; Dutta et al. 2017; Hosgood et al. 2015; Kamal et al. 2016; Olopade et al. 2017; Pollard et al. 2014; Shan et al. 2014; Weinstein et al. 2017). We focus here on PM, primarily $PM_{2.5}$, because it is the pollutant most commonly measured in health studies — and the one for which a wide range of previous work exists, characterizing both exposure and the health effects of exposure.

$PM_{2.5}$ measurements are typically characterized as either *integrated* or *real-time*. Integrated samples for $PM_{2.5}$ involve using a pump to draw air through a size-selective inlet (ensuring that the particles collected are approximately $PM_{2.5}$) and then depositing the selected particles on a filter. The filter is weighed before and after sampling, enabling estimation of the mass collected. The pump's run-time and flow rate can be used to calculate the volume of air sampled, allowing a concentration in mass-of-pollutant per volume air to be calculated. This type of sampling — known as gravimetric sampling — is considered the gold standard for $PM_{2.5}$ monitoring; however, the equipment (pumps, filters, and size selective inlets) are typically relatively heavy (due to large batteries) and noisy. Filter weighing requires sophisticated microbalances, and filter handling is ideally performed in near-clean room conditions. These constraints make gravimetric personal exposure assessment difficult in contexts with high solid fuel use rates. As an alternative, several real-time $PM_{2.5}$ monitors have been developed. These monitors typically use light scattering to measure particle concentrations and provide data at a user-defined time-interval, typically one minute. These monitors can be active (sampled with a pump) or passive and are typically small, lightweight, and quiet. They require regular calibration using a gravimetric sampler against the aerosol of interest for an accurate interpretation of data.

Personal exposure assessment is generally considered the most accurate and direct measurement technique. When assessing personal exposure, a monitor is placed near the breathing zone of the primary cook and is carried by her throughout her daily activities. Personal exposure assessment requires monitors that optimize between a number of competing factors — they need to be lightweight, quiet, battery-operated, and impose minimal discomfort on participants; they simultaneously need to be rugged and relatively inexpensive. Until recently, few monitors meeting these criteria have been available; as a result, personal monitoring has been fairly limited in scope. For example, a recent review of the effectiveness of stove interventions identified only 6 studies providing personal estimates of $PM_{2.5}$ out of 42 studies reviewed (Pope et al. 2017).

In late 2016 and early 2017, two new PM exposure monitoring samplers were released — the Enhanced Children's Micropem (ECM; Research Triangle Institute) and the Ultrasonic Personal Aerosol Sampler (UPAS; Access Sensor Technology). Both devices are fairly rugged, small, lightweight, quiet, and take advantage of modern microelectronics to measure a number of parameters related to HAP exposure. When fully validated, they may facilitate more rapid and easy personal exposure assessment.

An alternative to personal exposure assessment is microenvironmental monitoring and exposure reconstruction, which involves placing air pollution monitors in places in the house that the participant frequents — for instance, monitors may be placed in the kitchen, the living room, and a courtyard. Exposures can be reconstructed by coupling these stationary monitors with time–activity diaries or questionnaires that assess time spent in specific rooms or by using sensors that indicate the presence or absence of an individual in a room.

Existing Exposure Assessment Data

A number of recent reviews collect HAP measurement data from around the globe (a summary was introduced in Figure 3). WHO hosts the *global database of HAP measurements*, a comprehensive spreadsheet of global measurements last updated in 2011 (and currently undergoing revision). More recently, two reviews described the impact of interventions on HAP exposures and concentrations (Pope et al. 2017; Quansah et al. 2017). Both note a relative paucity of personal exposure data. Pope and colleagues (2017) reported pre-intervention mean exposures of 220 $\mu g/m^3$, approximately 6 times higher than the WHO Interim Target-1 of 35 $\mu g/m^3$. Mean post-intervention exposures across a range of intervention types (including improved biomass stoves with and without chimneys, LPG, and ethanol) were approximately 100 $\mu g/m^3$ or 2.8 times higher than the WHO Interim Target. These results are discussed in more detail in the section of the report "Intervention Exposure Studies."

Challenges and Uncertainty in Measuring Exposure

Concentrations and exposures to pollutants of interest are typically measured in intervals of 24 hours, allowing calculation of a 24-hour average comparable with global PM and CO guidelines. Because of the challenges of field measurement campaigns, however, little work has been done to characterize how representative these samples are. That is, how accurately does a single 24-hour or 48-hour

measurement predict an annual or multiyear exposure? Existing evidence indicates that within-subject variability exceeds between-subject variability (Dionisio et al. 2012; McCracken et al. 2009), demonstrating a need to understand how well short-term exposure measures predict long-term averages. This is especially relevant for understanding the relationships between exposure and chronic disease outcomes, which require estimates of multiyear or lifetime exposures. Variability can have a profound implication on the shape of exposure–response curves and their interpretation. Figure 20 highlights the uncertainty in both the accepted $PM_{2.5}$ IER curve for LRI and in pre- and post-intervention exposure measurements (vertical lines).

MODELING THE HEALTH BENEFITS OF REDUCED EXPOSURES

In recent years — driven in large part by a growing awareness of the impact of air pollution on health — there has been interest in and development of tools to estimate the potential health impacts of interventions to reduce air pollution exposures. These tools exist in the form of standalone software or spreadsheet-based models and mask complexity under an easy-to-use interface. They have been used to estimate the impact of policies and programs to reduce ambient air pollution and HAP and to model counterfactual scenarios in which air pollution exposures are driven down at subnational, national, and global levels.

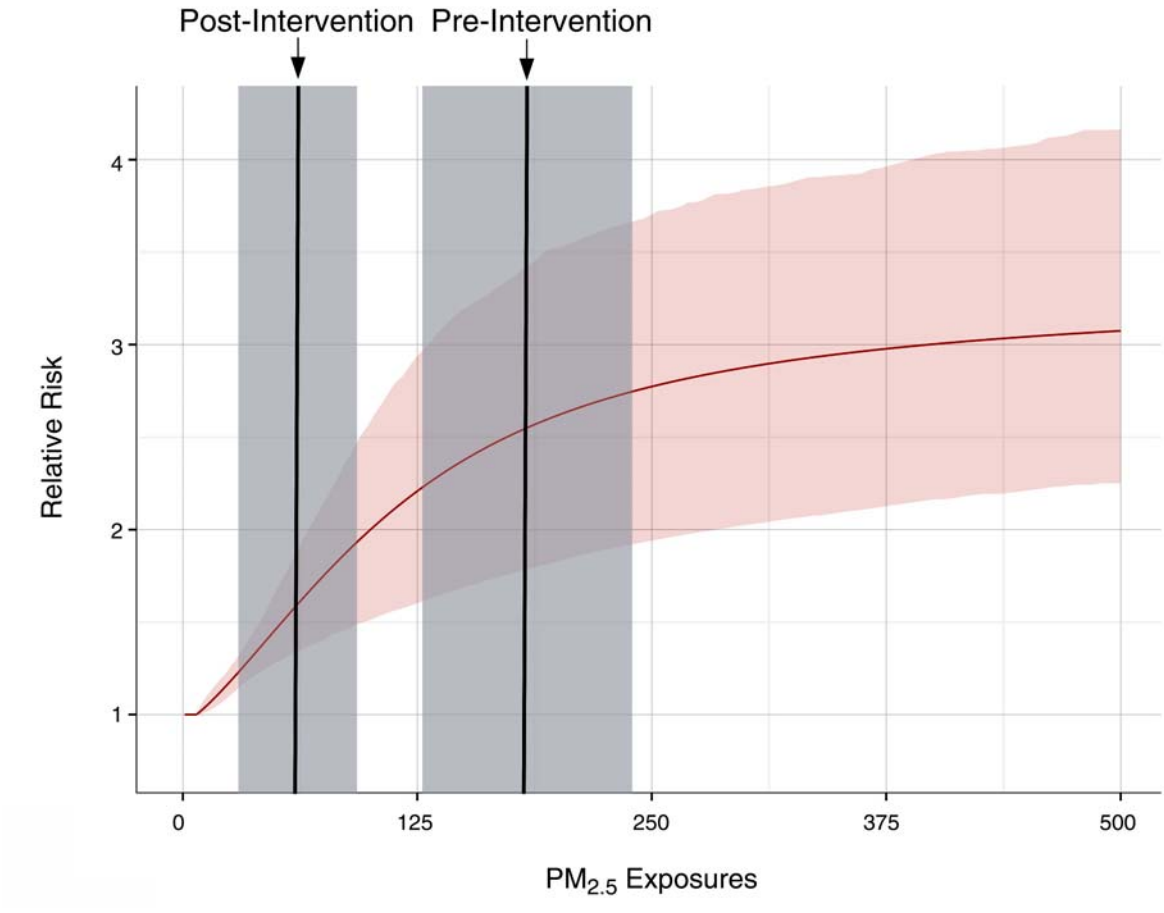

Figure 20. Integrated exposure–response curve for LRI with uncertainties. The red line is the exposure–response curve; the pink shading indicates the 95th percentile uncertainty bounds around the curve. The vertical black lines indicate hypothetical pre- and post-intervention exposures and measurement uncertainties (shaded gray). Data source: IHME.

Many of these tools rely on epidemiological evidence relating air pollution concentrations and exposures with health effects and use an attributable burden method to estimate changes in health status at the population level. While the fundamental approach between these models is similar, the underlying data can vary widely; some models utilize the IHME GBD integrated-response functions, while other functions derived from individual air pollution studies, such as those conducted in the American Cancer Society cohort (e.g., Krewski et al. 2009). Background disease data used to calculate metrics of interest — such as averted DALYs or deaths — vary among software tools and are often based on complicated, modeled estimates.

Anenberg and colleagues (2016) reviewed 12 such tools, noting that such software for HAP was at a relatively early stage of development. The remainder of this section will describe HAPIT, the Household Air Pollution Intervention Tool, a web-based software tool developed by University of California, Berkeley, with support from the Global Alliance for Clean Cookstoves.

The Household Air Pollution Intervention Tool

HAPIT estimates the health benefits attributable to household energy intervention programs that reduce exposure to HAP resulting from *dirty fuel use* (coal, wood, charcoal, dung, or other biomass). HAPIT uses (1) national data for 104 countries and subnational data for China and Mexico that are retrieved from IHME, (2) dirty fuel use data derived from the WHO, (3) population and demographic data retrieved from the Global Alliance for Clean Cookstoves, IHME, and publicly available census databases, and (4) lookup tables created for disease endpoints related to HAP following the example of Burnett and colleagues (2014) and using data available from IHME. HAPIT requires users to input parameters related to the chosen intervention, including the number of households that receive an intervention and the pre- and post-intervention $PM_{2.5}$ exposures.

HAPIT calculates averted health impacts in terms of DALYs, a combined metric of morbidity and mortality, and deaths; it focuses on the five diseases that are considered by GBD to have high-quality evidence: COPD, stroke, IHD, and lung cancer in adults and acute LRIs in children under 5 years old. Appendix C contains additional information on HAPIT methodology, future directions for HAPIT, and on integration with other software packages.

Modeled Health Benefits of Reduced HAP

A number of studies model the potential health benefits of adopting clean cooking technologies based either on assumed or measured exposure reductions. Such analyses are useful policy planning tools. When coupled with cost–benefit analyses like those described in section *Economic Burden of Household Air Pollution*, they enable comparisons of interventions to reduce HAP with other subnational, national, or regional health promotion strategies (for example, in terms of dollars spent per DALY/death avoided).

Prior to the development of the IERs, studies utilized point estimates of RR and reductions in emissions or exposure to evaluate potential health benefits. These papers used older estimates of underlying disease burden, older RR estimates, and relied on assumptions that no longer represent the state of the science. For example, in Mehta and Shahpar (2004) five intervention scenarios were considered for solid fuel using populations: a full transition (1) to LPG (no exposure), (2) to kerosene (no exposure), or (3) to improved stoves (exposures reduced by 75%); and a partial transition (4) to LPG and improved stoves or (5) to kerosene and improved stoves, in which 50% of the population experienced no exposure and 45% have their exposure reduced by 75%. The authors estimated that transitions to LPG or kerosene (still considered a clean fuel at the time of publication) would have the largest benefits in terms of gains in healthy life years, but that despite more modest exposure reductions, improved stoves would still have substantial potential for providing health benefits. Similar analyses have been performed for China (Aunan et al. 2013), Kenya (Malla et al. 2011), Nepal (Pant 2012), Nigeria (Isihak et al. 2012), India (Patel et al. 2016), and the Western Pacific Region (Arcenas et al. 2010). More recently, researchers have taken advantage of the continuous IER functions to estimate health benefits of interventions. Such analyses have been performed for Guatemala (Pillarisetti et al. 2016), Laos (Hill et al. 2015), Cambodia (Berkeley Air Monitoring Group 2015), and Mozambique (Anenberg et al. 2017). In all cases, the interventions programs were projected to be either cost-effective or very cost-effective. These types of modeling exercises highlight a tension between the apparent cost-effectiveness of interventions and the avoidable burden left unaddressed by interventions that do not decrease exposures substantially.

EVIDENCE FROM RECENT FIELD INTERVENTION STUDIES

Over the last 15 years, a number of researchers have carried out experimental clean cooking interventions and

have quantified the resulting exposure reductions and, in some cases, the health benefits. Overall, the results have been somewhat disappointing. In this section we review the evidence, and in the next section we consider some of the reasons why clean cookstove interventions have not delivered the expected improvements in exposure and health.

Intervention Exposure Studies

Pope and colleagues (2017) carried out a systematic review of studies that quantify reductions in kitchen HAP concentrations and in personal HAP exposures attributable to cookstove interventions, which ranged from low cost rocket stoves to clean fuels (LPG, ethanol, and electricity). They only included studies that assessed cookstove effectiveness in real-life use situations and that used valid exposure-assessment techniques. Most interventions — and in particular clean fuels interventions — delivered large percentage reductions in HAP levels, ranging from a 41% reduction in $PM_{2.5}$ kitchen concentrations for advanced combustion biomass cookstoves to an 83% reduction in kitchen concentrations for ethanol cookstoves. Given the large starting concentrations, however, these reductions generally failed to bring concentrations close to the WHO Interim Target-1 for an annual $PM_{2.5}$ of 35 μg/m^3 (Bruce et al. 2015b). Additionally, the number of studies is small. Only 42 studies were eligible for inclusion, over half of which (n = 23) evaluated kitchen concentrations resulting from chimney stove interventions. Only three studies have quantified kitchen concentration reductions from interventions using LPG and ethanol, clean fuels that are generally regarded as most promising, and no studies have yet reported changes in personal exposure resulting from clean fuels interventions. Finally, the review authors note that the exposure assessments used a highly heterogeneous set of assessment methods. The lack of a consensus method makes it hard to compare across studies.

Intervention Studies Excluding Randomized Control Trials

Observational studies, including cross-sectional and pre–post designs, can provide valuable information about the potential for different types of cookstove interventions in different locations to improve health status (Peel et al. 2015). In 2014, the WHO reviewed experimental and observational studies reporting on the health impacts of interventions (WHO 2014b). There were three studies examining the effects of improved stoves on acute LRIs (in Guatemala, China, India), three for adult respiratory health/COPD (Mexico, Guatemala, China), one for birth weight (Guatemala), and one for lung cancer (China). Only one of these, the RESPIRE trial in Guatemala, was a randomized control trial. None of the studies addressed cardiovascular effects.

The majority of the reviewed studies observed reduced HAP exposures and improved health outcomes. One study found that the reduced emissions observed in laboratory tests were not translated into exposure reductions in practice, and no health benefits were found (Hanna et al. 2016). The study authors recommended that intervention technologies should be tested in real-world settings prior to full-scale dissemination as households used the stoves irregularly and inappropriately, failed to maintain them, and reduced their usage of them over time. This finding is consistent with conclusions made elsewhere that intervention technologies should be piloted within the community prior to use in the intervention to increase the likelihood of adoption and use (Gold Standard Foundation 2017; Smith KR et al. 2015).

Based on these results, the WHO review (2014b) found that few, if any, interventions could achieve levels of $PM_{2.5}$ in the home that were even close to the WHO Interim Target 1 of 35 μg/m^3, and none could meet the WHO Air Quality Guideline of 10 μg/m^3. This result was observed for both solid fuel interventions and clean-burning gas stoves, which were likely ineffective at reducing exposures to WHO targets owing to ongoing use of both old and new fuels and stoves and continued pollution from neighbors and other sources. The WHO concluded that to meet the air quality guidelines, interventions should support the adoption of clean fuels for all purposes (including cooking, heating, lighting and other applications) across communities and as rapidly as is feasible.

Since the WHO review, another systematic review of the literature on the health benefits of cleaner fuels and solid fuel cookstoves, published through December 2015, was undertaken by Quansah and colleagues (2017). Of the 29 studies reviewed, 10 reported on respiratory health outcomes alone, 10 on nonrespiratory health outcomes, and eight on both respiratory and nonrespiratory health outcomes. While some studies observed improvements in health status with various types of interventions, others did not. The authors concluded that stand-alone HAP interventions yield little if any health benefit, and that there is a need to re-examine the ways in which interventions are designed and implemented in homes in low- and middle-income countries. While they note that cleaner fuels such as LPG, ethanol, solar, and electrification have the potential to substantially reduce exposures, only five of the studies evaluated impacts of cleaner fuels on health.

Table 11 summarizes studies published since the 2015 cutoff date used by Quansah and colleagues (2017) and some additional studies prior to that cutoff that appear not to have been included in previous reviews. These studies, conducted in India, Bolivia, China, and the Philippines, found reduced respiratory, cardiovascular, and ocular effects with the use of improved solid fuel cookstoves or cleaner fuels. However, study designs varied, some sample sizes were small, and the health effects measured were generally intermediate outcomes (e.g., blood pressure, lung function) as opposed to the incidence of cardiovascular and respiratory disease. Nevertheless, this newer evidence indicates that cleaner fuels and improved solid fuel stoves could lead to improved health status. Such results need to be confirmed with prospective intervention studies.

Randomized Control Trials of Stove and Fuel Interventions

Randomized control trials are typically considered the strongest study design in epidemiology for evaluating the causal effect of a treatment or intervention on a health outcome, owing to the similarities of treatment and control groups for all measured and unmeasured factors other than the assigned treatment. For example, because clean cookstove use is highly correlated with wealth in some settings, and because wealth is in turn often correlated with health outcomes of interest through causal channels that have nothing to do with air pollution, studies using fuel use as an exposure metric that do not randomize are likely to provide biased estimates of HAP risks and the benefits of interventions.

To date, four randomized control trials that investigate the effects of cookstove interventions on health outcomes have reported results (see Table 12 for a summary). Two additional trials have completed participant follow up and manuscripts are in preparation:

- The GRAPHS trial in Ghana recruited pregnant women into control, LPG, and forced-draft improved biomass groups and tracked birthweight and early childhood pneumonia (Jack et al. 2015).
- A trial in Nepal followed a similar design, recruiting women in early pregnancy; delivering both LPG and improved biomass interventions and tracking birthweight and pneumonia (Tielsch et al. 2014).

Finally, a large National Institutes of Health (NIH) and Gates Foundation multicenter trial is in its initial stages (*www.hapintrial.org*) (NIH Office of Extramural Research 2015).

Three things are striking about these trials. First, none have reported measured $PM_{2.5}$ reductions, even though $PM_{2.5}$ is generally considered to be the most important determinant of HAP health effects. Second, those that do report exposure assessment data have found reductions of about 50% for CO. These results are consistent with findings from studies of interventions' impact on exposure (in the earlier section "Intervention Exposure Studies") that current approaches to clean cooking appear not to deliver hoped-for exposure reductions. Third, in the case of the four randomized control trials summarized in Table 12, only the Nigerian trial delivered a clean fuel intervention. Unfortunately, no exposure data from that study have yet been published. The Orissa trial delivered a locally-manufactured cookstove that had not undergone rigorous pretesting (Hanna et al. 2016), and investigators studying the intervention stove in the Malawi trial found that it did not significantly reduce emissions factors under field conditions relative to traditional cookstoves (Wathore et al. 2017). The Plancha intervention stove in the RESPIRE trial reduced kitchen concentrations of CO by 90%, but reduced personal exposures by only 50% (Smith KR et al. 2011).

CHALLENGES IN REDUCING THE HAP BURDEN OF DISEASE

Why have cookstove interventions largely failed to deliver the exposure reductions that would be necessary to achieve the health benefits expected given the high burden of disease attributable to HAP? None of the exposure assessments described in the previous section were designed to address this question empirically. Through field observations and inferences from studies that track stove use over time, three factors have emerged that may help explain the observed high post-intervention exposures:

- *Low levels of adoption and sustained use*. Many cookstove programs have failed because households decline to adopt the intervention technology, or because they abandon use shortly after adoption (Bensch and Peters 2015; Pillarisetti et al. 2014; Ruiz-Mercado et al. 2011).
- *Stove stacking*. Even if households use clean stoves continuously, they may continue to use polluting solid fuel cookstoves for some cooking tasks. This has been well documented in field studies (Pillarisetti et al. 2014; Ruiz-Mercado et al. 2011).
- *Community-level exposures*. Neighborhood sources of pollution (including both nearby households that continue to use solid fuels and other combustion sources such as rubbish and agricultural burning) may result in exposure levels that exceed WHO targets. This has

Table 11. Recent Studies Evaluating Health Benefits of Cleaner Fuels or Solid-Fuel Cookstoves[a]

Author / Date / Country	Health Outcome	Methods	Results
Alexander et al. 2014, Bolivia			
	Cardiovascular — blood pressure	Pre–post study of 28 women using wood fuel for cooking and heating. Intervention included improved wood-burning cookstove, a chimney, and metal roof for kitchen. Measured household exposure and blood pressure prior to the intervention and 1 year after the intervention.	Significant decreases in systolic blood pressure and insignificant decreases in diastolic blood pressure with use of the intervention. Systolic blood pressure decreases were correlated with reductions in 24-hour mean kitchen $PM_{2.5}$ levels.
Capuno et al. 2016, Philippines			
	Respiratory, child — severe coughing with difficulty breathing	Propensity scores — Logistic regression on a data set comprising 5,442 children < 5 yrs. Matched with a control child (nearest neighbor). The counterfactual for the treatment child is another child who did not receive the treatment but otherwise had very similar characteristics as reflected in the propensity scores.	Incidence of severe coughing with difficulty in breathing lower by 2.4 percentage points for young children in households that use electricity, LPG, natural gas, or biogas than for controls that had biomass, kerosene, or solid fuel stoves in their homes.
Cheng et al. 2015, China			
	Respiratory — lung function; Eye symptoms — tearing, sore eyes, red eyes	371 rural households selected to participate. Of 371, 8 were selected to conduct IAP sampling. 413 women completed a questionnaire and 49 took part in lung function tests. Selected households received stove improvement and behavior modification training.	Insignificant improvement in percentage of predicted value of FEV_1 and FVC. Significant reductions in self-reported tearing, sore eyes, red eyes, phlegm, and fever.

(Table continues next page)

IAP = indoor air pollution; FVC = forced vital capacity; PAH = polycyclic aromatic hydrocarbon; PEFR = peak expiratory flow rate

[a] Excluding randomized control trials.

been observed in field settings (Balakrishnan et al. 2015a) and is also proposed by Pope and colleagues (2017) as the probable explanation for the poor field performance of clean fuel interventions.

The systematic underperformance of clean cookstove interventions has critical implications for the design of clean cooking programs and policies. Simply providing households with access to clean cookstoves and fuels will almost surely fail to achieve exposure reductions necessary to reduce health burden substantially. To achieve health goals, clean cookstove programs must address the triple-threat of low adoption, stove stacking, and community exposure. Devising strategies to address this triple threat should be a top priority for researchers and program implementers. The academic literature to date is largely silent on what might work. This is a key area for future research.

Even if cookstove interventions were able to achieve low exposure levels, it could be difficult to observe a signal in terms of avoided HAP-related health impacts for several reasons. First, chronic diseases associated with HAP exposure often take years to manifest. They can also take years to reverse, if ever, after exposures are reduced. These dynamics are difficult to observe in short-term studies typical of most HAP studies, although intermediate

Table 11 (*Continued*) Recent Studies Evaluating Health Benefits of Cleaner Fuels or Solid-Fuel Cookstoves[a]

Author / Date / Country	Health Outcome	Methods	Results
Lewis et al. 2017, India			
	Cardiovascular — systolic and diastolic blood pressure; Respiratory — lung function and number of days in the hospital for acute respiratory illness	Cross-sectional observational cohort of 105 households that use either traditional mud stoves or improved cookstoves, surveys, environmental air sampling, and health measurements.	Improved cookstove use was associated with 72% reduction in $PM_{2.5}$, 78% reduction in PAH levels, reduced time in the hospital with acute respiratory infection and reduced diastolic blood pressure.
Sukhsohale et al. 2013, India			
	Respiratory — lung function	Cross-sectional study in 760 non-smoking rural women cooking with biomass, kerosene stoves, LPG, and mixed fuels. Measured peak expiratory flow rate (PEFR).	Greater predominance of abnormal PEFR among biomass users compared with kerosene, LPG, and mixed fuel users.
Zhou et al. 2014, China			
	Respiratory — lung function and COPD	9-year prospective cohort study among 996 participants >40 years old from 2002 to 2011 in 12 villages in southern China. Interventions included improving kitchen ventilation and promoting use of biogas, interviews, pollutant monitoring, spirometry tests.	Use of clean fuels and improved ventilation were associated with a reduced decline in FEV_1 and risk of COPD. Benefits were greater for combined clean fuels and improved ventilation, and for longer duration of both.

IAP = indoor air pollution; FVC = forced vital capacity; PAH = polycyclic aromatic hydrocarbon; PEFR = peak expiratory flow rate

[a] Excluding randomized control trials.

outcomes (e.g., blood pressure) can be observed and may provide indications as to the development of long-term diseases. Another complicating factor in the observability of health benefits from reduced HAP exposures is that cookstove interventions often influence other determinants of health, including diet, physical activity, and other lifestyle changes. Such changes in individual level risk factors are interrelated (Kurti et al. 2016), but to date have not been sufficiently explored to allow an understanding of the full consequences of cookstove interventions. Finally, population dynamics, including population growth and aging, may have more prominent impacts on NCDs than HAP exposures, and thus obscure the signal from the change in HAP exposure alone (Bonjour et al. 2013; Cohen et al. 2017, World Bank and IHME 2016; WHO 2014b). These challenges do not mean that reduced HAP would have no health benefits — our understanding of the exposure–response relationships indicate that reduced HAP exposures should result in reduced risk of respiratory disease, lung cancer, cardiovascular disease, cataracts, and other adverse health outcomes absent changes in other risk factors attributable to the intervention. These risk reductions might, however, be difficult to detect even with substantial observed HAP exposure reductions.

Table 12. Summary of Randomized Control Trials Investigating the Effects Of Cookstove Interventions on Health Outcomes

Location Primary Outcome Citation	Design	Results	Exposure Reduction
Guatemala (RESPIRE) Pneumonia Smith KR et al. 2010, 2011	• Household level randomization • Chimney cookstove delivered to rural households with pregnant woman or young child • Total *n* = 518 households	Significant reduction in fieldworker-assessed, physician-diagnosed, and respiratory syncytial virus negative pneumonia	Personal CO assessed via diffusion tubes. Kitchen CO concentrations reduced by 90% and personal exposures reduced by 50%
Orissa, India Adult lung function Hanna et al. 2016	• Household level randomization • Very low cost clay chimney stove delivered in three waves to rural households • Total *n* = 2,651 households	No evidence of lung function benefits	Assessed via exhaled CO[a]. Significant reduction in first 12 months post intervention, no reduction thereafter
Nigeria Blood pressure and biomarkers in pregnant women Alexander et al. 2017 Northcross et al. 2016 Olopade et al. 2017	• Household level randomization • Ethanol cookstoves and fuel delivery to urban kerosene and woodfuel users • Total *n* = 324 households.	Significant reduction in diastolic blood pressure in pregnant women (Alexander et al. 2017) Limited evidence that women who used wood fuels at baseline experienced reduction in an inflammatory biomarker (TNF-α) (Olopade et al. 2017).	Not yet published
Malawi (Cooking and Pneumonia Study) Child acute lower respiratory infection Mortimer et al. 2017	• Community-level randomization with household level intervention[b] • Intervention was a forced-draft biomass stove • Total *n* = 10,750	No significant reduction in pneumonia risk	50% reduction in child personal CO exposures; exposure assessment methodologies have not been published

[a] Exhaled CO is a valid biomarker of recent exposure.

[b] Only households with young children were eligible to receive the intervention stove.

CONCLUSIONS

This report provides an updated literature review on the health effects of HAP exposure, with particular emphasis on NCDs in low- and middle-income countries. *Widespread use of solid fuel stoves by approximately one third of the world's population imposes a heavy burden on global public health. The most recent estimate from the IHME GBD study estimates that in 2016, the number of deaths attributable to HAP was 2.6 million worldwide, making it the 8th leading risk factor globally, and with ambient air pollution, the leading environmental risk factor.* NCDs account for approximately three-quarters of the deaths attributable to HAP globally. The disease burden attributable to HAP remains largest in South Asia and sub-Saharan Africa.

Given the contribution of NCDs to the current estimates of the HAP burden of disease, it is important to understand the strength of the evidence underlying the associations between HAP and each of these health outcomes. The objective of this report was to examine the basis for much of the recent estimates of the burden of HAP exposure on NCDs, to identify the key uncertainties in the state of the knowledge as recently reviewed comprehensively by the WHO, IHME, and other organizations, and to evaluate the extent to which the most recent literature has addressed those uncertainties.

Overall, the new evidence reviewed in this report is broadly consistent with previous conclusions that HAP is strongly associated with numerous diseases. A large body of epidemiological evidence — including multiple systematic reviews, but few experimental studies — suggests associations of HAP exposure with COPD. New, large studies provide contradictory evidence on these associations, limited by reliance on proxy-based indicators of exposure (e.g., fuel use) among other factors. A smaller but growing body of cross-sectional and case–control studies link childhood asthma to HAP exposures, but further research is needed to fully understand the association. Inference from studies of ambient air pollution and cigarette smoking indicate that HAP is also likely associated with cardiovascular disease, although until recently there has been relatively little direct evidence from HAP studies. Recent results, especially a large cohort study from Iran (Mitter et al. 2016), corroborate cardiovascular risk estimates for a specific household fuel (kerosene), but not biomass; a large cohort study in China found higher risks of cardiovascular mortality among those reporting use of solid fuels for cooking and heating. Studies that link HAP exposure to higher blood pressure provide indirect evidence of cardiovascular risk.

Building on strong evidence for a link between household coal emissions and lung cancer, summarized in IARC's classification of smoke from indoor coal burning as a human carcinogen, recent work has strengthened the evidence for a link between lung cancer and biomass cooking smoke. Findings from one recent study suggest there could be a dose–response relationship for years of exposure to biomass-based HAP and lung cancer. Relatively strong evidence links HAP with cataracts in South Asia, including a review by the WHO concluding that there is a reasonable case for causality, but some potential confounding factors (such as diabetes and ultraviolet light exposure) have not been adequately addressed in the literature. Additional evidence indicates potential associations between HAP exposure and other health outcomes, including pregnancy and birth outcomes, neurological and cognitive conditions, and diabetes; however, evidence remains limited and is still largely dependent on studies done in developed-country contexts.

There are three main weaknesses that create knowledge gaps and challenges in the existing HAP epidemiological literature. First, personal exposure to HAP has largely been classified based on use of solid fuels, rather than on direct measurement of exposure to study participants. These categorical measures increase the possibility of exposure misclassification and cannot be used to understand the quantitative relationships between exposure to $PM_{2.5}$ and other HAP components. A related exposure issue is that the development of NCDs is often a function of decades of exposure to HAP, which means that even careful exposure assessment during a short study period may not be sufficient to characterize longer-term exposure. Second, many studies use imprecise measures to characterize health outcomes; for example, relying on questionnaires, rather than validated biological or physiological measures such as spirometry. Third, solid fuel use is closely related to poverty and other known risk factors for NCDs, which are not rigorously taken into account in many observational studies. Few studies have been carried out in such a way as to allow causal inference, by accounting for the fact that solid fuel users and cleaner fuel users are likely to differ in other ways that are relevant to health outcomes under study.

While these weaknesses reduce confidence in the current body of epidemiological evidence, they also highlight opportunities for further research to strengthen the evidence base. Determining the risk of health outcomes with long latency between exposure and outcome requires study designs that minimize confounding and carefully assess exposure, including both long-term observational studies, randomized control trials, and relatively inexpensive and fast case–control studies. High-quality, targeted

studies that focus on outcomes of interest are necessary to strengthen the evidence base linking HAP exposure with NCDs. While strong evidence supports associations of cardiovascular disease with ambient PM and cigarette smoking, and more recently with solid fuel use in the home, such relationships have yet to be demonstrated for more specific measures of HAP exposures. Additional research is also needed to strengthen the evidence base for understanding the associations between HAP and respiratory diseases (e.g., COPD and asthma), cancers other than lung cancer and cancer pathogenesis, and cataracts and other eye diseases in populations other than women in South Asia. It also remains unclear whether different pollution mixtures have stronger or weaker associations with each health outcome. For example, how do particles from coal combustion compare with particles from wood combustion? The WHO has strongly recommended against the use of unprocessed coal for household energy, because coal contains toxic elements (e.g., fluorine, arsenic, lead, selenium, and mercury) that are not destroyed by combustion and because there appear to be technical constraints on burning coal cleanly in households (WHO 2014b). Similarly, are particles from biomass pellets less toxic than particles from unprocessed wood and agricultural waste, and other fuels that continue to be used, such as animal dung and trash? Ongoing scientific inquiry may shed light on these questions over time. How do these uncertainties affect the shape of the exposure–response relationships for different exposures and populations, which have implications for the level of reductions that would be needed to achieve meaningful health benefits?

IER models based on evidence from exposure to ambient air pollution, HAP, environmental tobacco smoke, and cigarette smoking now make it possible to estimate the current health burden of HAP, as well as the potential health benefits of reducing HAP for any population of the world for which exposure levels are known or can be estimated. However, several recent household-level solid fuel stove randomized control trials and observational studies have delivered less-than-expected reductions in HAP exposures and improvements in health. Some technologies have proven that they can reduce emissions in both the laboratory and the field, but no currently available technology has proven that it can reduce exposures to the levels of the WHO Air Quality Guidelines for $PM_{2.5}$ (or even the less stringent interim targets). Giving a household new cooking technology does not ensure that the technology will be consistently used or that its use will be enough to overcome other sources of exposure. Such households may continue using the traditional stoves and fuels. Continued exposure from exposures outside the home — including the use of traditional stoves and fuel by others in the community, as well as from traffic, trash burning, and agricultural burning — may also obscure any benefits. The disappointing findings may also be driven by similar factors that challenge epidemiological studies, among them the challenges of studying chronic disease outcomes with shorter term studies. Based on the shape of the IERs, measurable health benefits would not be expected without substantial exposure reductions to low levels at or below the WHO guidelines. Even if interventions substantially reduce exposure levels, the interventions may lead to additional changes in lifestyle, diet, activity levels, and other factors that can also influence health status. More broadly, demographic changes such as population growth and aging may increase the numbers of people susceptible to NCDs in general, thus obscuring the signal from the change in HAP exposure alone (Bonjour et al. 2013; Cohen et al. 2017; World Bank and IHME 2016; WHO 2014b).

Though data gaps and challenges in intervention effectiveness remain, epidemiological evidence indicates that reducing HAP exposures should be an effective way to improve public health worldwide. With substantial reductions in real-world exposure levels, reductions in the NCDs that have been associated with HAP exposures — as well as from a number of emerging adverse health outcomes — would be expected. Given where household burning of solid fuels is most prevalent, these benefits would accrue largely in low- and middle-income countries to families and individuals living in poverty and at the bottom rungs of the energy ladder. Many interventions would also have additional co-benefits for society from improved ambient air quality and reduced impacts on both near-term and long-term climate change. Some published studies have estimated that the health benefits of reduced HAP exposures likely outweigh the costs of intervention.

Accelerating transitions to modern fuels and electricity that are most likely to achieve the necessary exposure reductions would be an ideal path forward. At the same time, access to modern fuels remains out of reach for many communities that rely on solid fuels for household energy needs. Improved solid fuel stoves that burn fuel more efficiently and reduce emissions are available, but data from the field suggest that possession of a biomass stove is not sufficient for reducing HAP exposure. Efforts to mitigate HAP should now address the economic and behavioral barriers to sustained adoption of clean stoves and fuels, simultaneously with other sources of combustion-related pollution in affected communities. Programs and strategies that provide scattered households with access to clean fuels — without addressing the challenge of sustained, exclusive use — are unlikely to deliver meaningful public

health benefits. *Strategic efforts are now needed to change, and perhaps transform, energy systems to deliver high-quality energy services to low-income households, not only for cooking, but also for heating and lighting.* Exploration of different approaches that will achieve sustained low air pollution exposures is still needed.

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APPENDIX A: LITERATURE SEARCH

We identified 18 previously published reviews of household air pollution and health outcomes, coded these according to the outcomes they addressed, and used those that reported quantitative risk values as a starting point for each health outcome. The methods are described in the section "Effects of Household Air Pollution on Noncommunicable Diseases."

Table A.1 provides a listing of the previous reviews of household air pollution and health outcomes used as a starting point for each section on associations between household air pollution and individual health outcomes.

The search terms and results of the literature search are presented in Table A.2. Date constraints (noted in right column) were based on the publication year of the last systematic review on the impact of household air pollution on the particular outcome.

Table A.1. Previous Reviews of HAP and Health Outcomes Referenced in This Report

Author	Year	Health Endpoints Addressed	Systematic Review Guidelines?	Assessed Heterogeneity or Publication Bias?
World Health Organization	2014b	Respiratory, cardiovascular, lung cancer, cataracts, birth outcomes.	Yes	Yes
Bruce et al.	2015a	Lung cancer	Yes	Yes
Hosgood et al.	2011	Lung cancer	Yes	Yes
Zhang and Smith	2007	Lung cancer, COPD	Not reported	Not reported
West et al.	2013	Cataracts	Not reported	Not reported
Smith KR et al.	2014	Respiratory, cardiovascular, lung cancer, cataracts, birth outcomes, diabetes	Yes	Yes
Kulkarni et al.	2014	Cataracts	Not reported	Yes
Pope et al.	2010	Birth outcomes	Yes	Yes
Bruce et al.	2013	Respiratory, birth outcomes	Yes	Yes
Amegah et al.	2014	Birth outcomes	Yes	Yes
Eisner et al.	2010	COPD	Yes	No
Hu et al.	2010	COPD	Yes	Yes
Kurmi et al.	2010	COPD	Yes	Yes
Po et al.	2011	COPD, asthma	Yes	Yes
Gordon et al.	2014	COPD, asthma	No	No
McCracken et al.	2012	CVD	No	No
Fatmi and Coggon	2016	CVD	No	No
Bruce et al.	2015b	All-cause mortality, respiratory, lung cancer, cancer of larynx, oro- and hypo-pharynx, cervical cancer, cataracts, birth outcomes	Yes	Yes

Note: COPD = chronic obstructive pulmonary disease; CVD = cardiovascular disease.

Table A.2. Search Terms and Results of the Literature Search for Studies on Associations Between Household Air Pollution and Health Outcomes

Search Terms	Search Engine	Number of Results: Not Date-Constrained	Number of Results: Date-Constrained
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND ("asthma"[MeSH Terms] OR "asthma"[All Fields])	PubMed	1,449	No date-constrained search
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: (asthma)	Web of Science	588	No date-constrained search
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "birth outcomes"[All Fields]	PubMed	11	5
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("birth outcomes")	Web of Science	278	245
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "blood pressure"[All Fields]	PubMed	45	25
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("blood pressure")	Web of Science	293	256
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "burns"[MeSH Terms]	PubMed	18	0
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("burns")	Web of Science	277	245
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "neoplasms"[MeSH Terms]	PubMed	613	56
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("cancer")	Web of Science	549	218
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "cardiovascular disease"[All Fields]	PubMed	42	12
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("cardiovascular disease")	Web of Science	300	173
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "cataract"[MeSH Terms]	PubMed	5	1
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "eye disease"[All Fields]		3	1
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "ocular disease"[All Fields]		0	0
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("ocular disease")	Web of Science	270	241
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "cervical cancer"[All Fields]	PubMed	2	No date-constrained search

(Table continues next page)

Table A.2 *(Continued).* Search Terms and Results of the Literature Search for Studies on Associations Between Household Air Pollution and Health Outcomes

Search Terms	Search Engine	Number of Results	
		Not Date-Constrained	Date-Constrained
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("cervical cancer")	Web of Science	276	No date-constrained search
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "cognitive" [All Fields]	PubMed	36	No date-constrained search
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: (cognitive)	Web of Science	280	No date-constrained search
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "pulmonary disease, chronic obstructive"[MeSH Terms]	PubMed	97	15
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: (COPD)	Web of Science	362	189
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "diabetes" [All Fields]	PubMed	17	9
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("diabetes")	Web of Science	281	246
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "wounds and injuries"[MeSH Terms]	PubMed	202	24
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("injuries")	Web of Science	278	214
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "neurological" [All Fields]	PubMed	30	No date-constrained search
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: (neurological)	Web of Science	275	No date-constrained search
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "pneumonia"[MeSH Terms]	PubMed	61	5
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: (pneumonia)	Web of Science	347	183
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND ("tuberculosis"[MeSH Terms] OR "tuberculosis"[All Fields])	PubMed	129	No date-constrained search
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: (tuberculosis)	Web of Science	338	No date-constrained search
("household air pollution"[All Fields] OR "indoor air pollution"[All Fields]) AND "violence" [MeSH Terms]	PubMed	39	7
("household air pollution") OR TOPIC: ("indoor air pollution") AND TOPIC: ("violence")	Web of Science	277	269

APPENDIX B: HOUSEHOLD AIR POLLUTION IMPACTS ON NEAR-TERM AND LONG-TERM CLIMATE CHANGE

Incomplete combustion of household fuels — such as wood, coal, dung, and kerosene — produces pollutants that contribute to climate change. These pollutants include black carbon, a potent short-lived forcer, and methane. Traditional solid fuel stoves and open cooking fires may account for over 1,500–1,700 Gg of black carbon annually, or about 20% of global black carbon emissions (Butt et al. 2016; Klimont et al. 2016; Putti et al. 2015); the upper range of this estimate was from a paper that considered only biomass emissions, so including coal emissions would increase the estimate. Also, household solid fuel heating emissions add an additional 4,000 Gg of black carbon annually (Klimont et al. 2016). Notably, cooking with solid fuels also releases organic carbon, which can have a variable or cooling effect on the global climate. Cooking with solid fuels may contribute 9,000 Gg of organic carbon per year (Klimont et al. 2016). In Asia and Africa, residential solid fuel use accounts for 60%–80% of black carbon emissions (WHO 2016). Kerosene burned for lighting is the source of 270 Gg of black carbon per year, contributing the warming equivalent of 240 million tons of carbon dioxide (CO_2) (WHO 2016).

The impact of household solid fuel use on CO_2 and other longer-lived climate pollutants is more complicated. It is clear that household use of coal results in CO_2 emissions. Some biomass used by households may be considered renewable, if deforestation is not occurring in the areas from which the fuel was harvested. However, if not regenerated over time, the use of biomass for household energy may contribute to CO_2 emissions. A report from the Global Alliance for Clean Cookstoves found that cooking with solid fuels, and related charcoal production, is estimated to generate 0.5–1.2 billion MT of CO_2 equivalent annually, or about 1.5%–3% of global CO_2 emissions (Putti et al. 2015). Another study estimated that burning wood for fuel accounts for more than one gigaton of CO_2 emissions per year, or about 2% of total global emissions (Bailis et al. 2015).

Several studies have concluded that reducing emissions from household fuel use by: (1) replacing traditional household solid fuel use with lower-emission cookstoves; (2) shifting to the use of cleaner fuels; and/or (3) improving building design or weatherization, can have multiple benefits for climate and health (WHO 2015b). Reducing ambient $PM_{2.5}$ from household cooking, in a hypothetical 20-year phase-out of cookstove emissions in each country with greater than 5% of the population using solid fuels for cooking, could have the potential to prevent 22.5 million premature deaths by the year 2100 (Lacey et al. 2017). Abatement in China, India, and Bangladesh would contribute to the largest reduction of premature deaths from ambient air pollution, preventing about 200,000 deaths per year (in year 2050) (Lacey et al. 2017).

A study of Mozambique found that, for rural areas, a 10% increase in the number of households using forced-draft wood-burning stoves could achieve >2.5 times more health benefits from reduced $PM_{2.5}$ exposure compared to natural-draft stoves in the same households, assuming 70% of households use the new technology for both cases (Anenberg et al. 2017). Interventions such as this could reduce expected climate change-related temperature increases from continued solid fuel use by 4%–6% over the next century.

A review focused on climate change impacts in the cryosphere found that emission reductions achieved by offering cleaner cookstoves or household fuels offer the greatest potential benefits both to human health and to slowing cryosphere warming. This review estimated that improving biomass and coal-heating stoves could save about 230,000 lives annually (Pearson et al. 2013). However, there is some evidence that some supposedly improved cookstoves used in interventions may release more black carbon per unit $PM_{2.5}$ than the emissions from pre-intervention cooking (Aung et al. 2016; Kar et al. 2012).

APPENDIX C: THE HOUSEHOLD AIR POLLUTION INTERVENTION TOOL (HAPIT)

HAPIT INPUTS AND DEVELOPMENT

HAPIT requires a number of user inputs, including the following:

- The country where the project is occurring (or the district of Mexico / province of China).
- The mean and standard deviation of the pre- and post-intervention $PM_{2.5}$ exposures. It is strongly suggested that measured personal exposure values be used as inputs to HAPIT.
- The number of households targeted by an intervention and the number of people, adults, and children per household.
- The fraction of households ever using the intervention and the intervention's lifetime.

Additionally, users can apply an adjustment factor to the exposure assigned to children and to adults who don't cook based on the exposure assigned to the primary cook.

HAPIT uses exposure inputs to recreate pre- and post-intervention exposure distributions and draws 1,000 pairs of values. For each set of these values, the risk of each disease at a given pollution exposure is estimated using the IERs. The relative risks are used to estimate the population-attributable fraction for each disease. The population-attributable fraction is the fraction of the background disease rate that is attributable to $PM_{2.5}$ pollution (rather than, say, high cholesterol intake). The difference between the disease attributable to $PM_{2.5}$ at the pre- and post- exposure levels is the benefit of the intervention. Burden-of-disease estimates and health benefits estimated by HAPIT require definition of an *ideal* counterfactual exposure, below which there is no risk to health. In the 2010 Burden of Disease, this value was set at 7.3 $\mu g/m^3$ for annual average $PM_{2.5}$ exposure. In HAPIT, the counterfactual is set at 7 $\mu g/m^3$. A complete description of HAPIT and its underlying methodology can be found in Pillarisetti and colleagues (2016) and in the Gold Standard Foundation's Methodology to estimate and verify averted disability-adjusted life-years from cleaner cooking and cleaner household air (Gold Standard Foundation 2017).

The initial version of HAPIT was created as a spreadsheet. In 2013, HAPIT was rebuilt using R, Shiny (a framework for bringing R programs to the web), C++, and JavaScript and updated to use the Global Burden of Disease (GBD) 2013 data from the Institute for Health Metrics and Evaluation. HAPIT is available online at *householdenergy. shinyapps.io/hapit3*.

HAPIT LIMITATIONS AND FUTURE DIRECTIONS

HAPIT has a number of limitations. First, it is constrained by changes in modeled IHME background disease data and changes in exposure–response functions. The currently available version of HAPIT uses 2013 GBD data; GBD data have been updated annually since 2015. Furthermore, the integrated exposure–response (IER) currently used in HAPIT is not the latest version. These changes underlie a more fundamental issue with this type of health-impact modeling; as background data and concentration–response functions change, estimates of averted ill health also change, sometimes dramatically. While this reflects the somewhat volatile state of the science, it can be difficult to convey dramatic changes in burden estimates to policymakers and nongovernmental organizations, who may rely on these estimates to secure funding. Second, HAPIT does not take into account uncertainty in the IERs. Doing so is not currently computationally tractable; as with all software tools, a compromise must be struck between technical diligence and ease-of-use. Third, HAPIT does not calculate potential community-level benefits of stove changeout programs that may impact local ambient air pollution levels and provide benefits to community members not necessarily involved in an intervention program. Implementation of such features is currently under consideration. Finally, HAPIT does not currently forecast changes in disease and death rates or population growth. To account for potentially significant changes in underlying disease burden, HAPIT limits the lifetime of an intervention to at most five years and encourages users to model potential benefits over even shorter timeframes.

ABBREVIATIONS AND OTHER TERMS

BOLD	Burden of Obstructive Lung Disease
CI	confidence interval
CO	carbon monoxide
CO_2	carbon dioxide
COPD	chronic obstructive pulmonary disease
DALY	disability-adjusted life-year
ETS	environmental tobacco smoke
FEV_1	forced expiratory volume during the first second
GBD	Global Burden of Disease (study)
GBD MAPS	Global Burden of Disease from Major Air Pollution Sources
HAP	household air pollution
HAPIT	Household Air Pollution Intervention Tool
HR	hazard ratio
IARC	International Agency for Research on Cancer
IEA	International Energy Agency
IER	integrated exposure–response
IHD	ischemic heart disease
IHME	Institute for Health Metrics and Evaluation
LPG	liquefied petroleum gas
LRI	lower-respiratory infection
NCD	noncommunicable disease
NIH	National Institutes of Health
OR	odds ratio
PM	particulate matter
$PM_{2.5}$	particulate matter ≤2.5 μm in aerodynamic diameter
PM_{10}	particulate matter ≤10 μm in aerodynamic diameter
RESPIRE	Randomized Exposure Study of Pollution Indoors and Respiratory Effects
RR	relative risk
SDI	sociodemographic index
VSL	value of a statistical life
WHO	World Health Organization

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